Manual of Military Ophthalmology

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MANUAL OF
MILITARY OPHTHALMOLOGY

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For the Use of Medical Officers of the Home, Indian, and Colonial Services

BY

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PREFACE.

I HAVE long thought that a small manual, containing not only instructions in vision-testing and the estimation of refractive errors, but also a concise account of the present state of knowledge of other ophthalmic subjects of special interest to Army surgeons and Colonial medical officers, such as eye injuries, malarial and venereal eye affections, malingering, the commoner eye operations, etc., would fill a gap in medical literature and prove of real service in military and tropical practice.

Since my return from active service in South Africa I have endeavoured during my scanty leisure to materialise this idea : this little volume is the outcome of the attempt.

A work of this kind, avowedly intended for beginners, can lay but little claim to originality save in its form and aim. I have endeavoured to bring together in compact form all, or nearly all, the ophthalmic subjects of special interest to military surgeons and practitioners in the tropics. Some of these are dealt with in the admirable manuals of Professor Fuchs, Mr. Hartridge, and Mr. Swanzy, works to which I hasten to express my indebtedness ; while others, notably the excellent descriptions of malarial eye diseases by Sulzer and Raynaud, and the researches into the etiology of trachoma by Chibret, are only to be found in scattered form in elaborate "Systems," monographs, and contri-

butions to the ophthalmic press of various countries, which the ordinary reader has neither the time nor the opportunity to consult.

I am indebted to the kindness of Professor Fuchs and Mr. Hartridge for permission to reproduce several diagrams from their works ; and to Mr. Stanford-Morton for an excellent and detailed account, with drawings, of the strabismus operation which he performs with such success. The drawings of eye instruments have been obtained through the courtesy of Messrs. Weiss.

No one is more painfully conscious than I am of my own deficiencies in undertaking this task. I can only appeal to my *confrères* to remember that its aim is a modest one, and that I do not pose as making an original or weighty contribution to ophthalmic literature.

CONTENTS.

MILITARY OPHTHALMOLOGY.

CHAPTER I.

THE OPTICAL PROPERTIES OF THE EYE.—THE USE OF LENSES.

The optical properties of the normal eye include refraction, accommodation, and convergence.

REFRACTION.

The word "refraction" is here used to mean the state of refractivity of the eye in a state of rest. The refractive surfaces and media of the normal eye act as

Fig. 1. (Hartridge.)

a convex lens with focus of 23 m.m. The divergent rays proceeding from an object seen are focussed by this convex lens on the retina so as to form a clear image (see diagram, Fig. 1).

A glance at the diagram shows that the image of the candle formed on the retina is inverted; why, then, is the candle seen upright? The explanation is that

we do not actually *see* the image on the retina ; the rays of light from the object seen produce certain sensations in the retina, and we *refer* these sensations to the object seen ; hence, *a* in the diagram is referred to A, *b* to B, *c* to C. Experience and the sense of touch

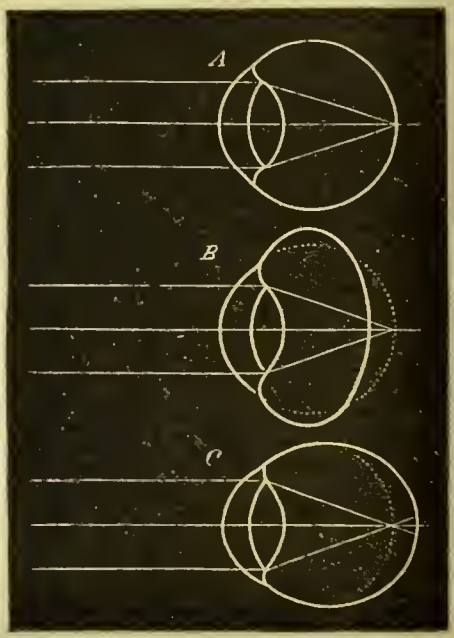


FIG. 2. (HARTRIDGE.)
A, Emmetropic eye. B, Hypermetropic eye. C, Myopic eye.

reinforce this process, with the result that the eye knows that the sensation felt in the upper part of the retina proceeds from the lower part of the object, and *vice versâ*.

In an emmetropic eye parallel rays of light (rays from a distant object diverge so slightly that we assume them to be parallel) are focussed *on* the retina, as the eye is of normal length ; in the myopic eye, which is too long, they are focussed *in front* of the retina ; in the

hypermetropic eye, which is too short, *behind* the retina (Fig. 2).

For the sake of simplicity I am now assuming that these errors of refraction are solely due to variations in the length of the eye.

In astigmatism the front of the eye—the cornea—is unequally curved, so that the refraction of opposite corneal meridians is different. A ready way of demonstrating astigmatism to a class is by slightly squeezing opposite sides of half a hollow rubber ball.

The most elementary acquaintance with optics will show that a concave lens, which diverges rays, will, if placed in front of a myopic eye, push the focus back to the retina; a convex, converging rays, will draw it forward to the retina in the hypermetropic eye; a combination of lenses to suit each meridian will be required in the astigmatic eye.

Errors of refraction in general are spoken of as ametropia. An expression not often used out of books is anisometropia, which means a variation in the refraction of the two eyes. Myopia and hypermetropia are known among the laity as "short sight" and "long sight" respectively. Emmetropia, of course, means normal vision.

The "visual angle," the importance of which will be seen in the next chapter, is the angle formed by rays proceeding from the extremities of an object at the nodal point of the eye—a point which for practical purposes may be considered to be the centre of the posterior surface of the lens.

In the diagram (Fig. 3) A B and C D are objects held at different distances from the eye; A N B and C N D are the visual angles formed by each. Obviously, the image formed at 1 (hypermetropic or shorter eye) will be smaller, at 3 (a myopic or longer eye) larger, than at 2 (the emmetropic or normal-length eye).

The expression "acuteness of vision" or "visual acuity" refers solely to the sensitiveness of the retina; an ametrope, if corrected by appropriate lenses, may therefore see as well as an emmetrope. In high forms of ametropia, however, the receptivity or sensibility of the retina (greatest at yellow spot, diminishing at peri-

phery) is liable to suffer. In the normal eye the retinal sensibility diminishes with advancing age.

ACCOMMODATION.

When we look at a distant hill through the branches of a tree near at hand we notice that the tree is not seen distinctly; on fixing our gaze on the tree the hill becomes indistinct: in fact, both objects cannot be focussed on the retina at the same time. The eye which has been looking at the hill must undergo a change of shape before it can see the tree distinctly, and *vice versâ;* in other words, it must *accommodate* itself to

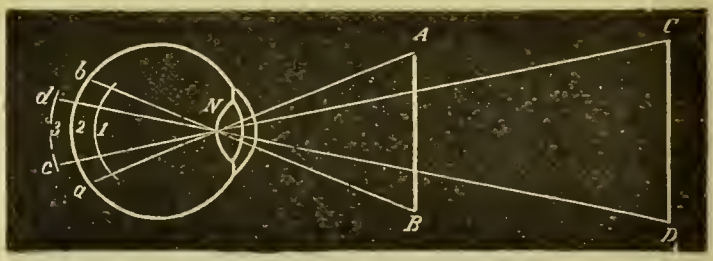


FIG. 3. (HARTRIDGE.)

varying distances. The power of accommodation depends on changes in shape of the lens produced by the action of the ciliary muscle. This muscle relaxes the capsule of the lens, and allows that elastic body, which has a natural tendency to become spherical when not pressed by the capsule, to become more convex in order to focus the diverging rays from near objects on the retina. The rays from distant objects are so slightly divergent that they may be considered parallel for practical purposes, and hence in emmetropes do not require any effort of accommodation (*i.e.* change in shape of lens by action of the ciliary muscle) to focus them on the retina.

The "range of accommodation" is between the far point (considered, for convenience, as any distance beyond 6 metres) and the near point, *i.e.* the point nearest the eye at which an object can still be distinctly seen. The near point gradually recedes with advancing

age, owing to diminution of the elasticity of the lens This diminution begins at the age of ten, and by the time forty-five is reached the emmetropic eye is no longer able to focus clearly objects nearer than 22 c.m., and glasses (convex) have to be worn for near work.

A little consideration, aided by the diagrams in Fig. 2, will show that the near point will recede earlier in life in a hypermetrope, later in a myope. In the case of the latter we can easily realise that with myopia beyond a certain amount these convex glasses for near work (glasses for presbyopia) need never be worn at all.

So far we have only spoken of *absolute* accommodation, or the accommodation of one eye. The normal human being has, however, two eyes, both participating in the act of vision; the act of accommodation carried out by both eyes simultaneously is known as *binocular* accommodation, and implies the power of *convergence.*

CONVERGENCE.

The so-called "visual line" is an imaginary line extending from the object looked at to the macula. Convergence is the power, exercised by the internal recti, of converging the visual lines to a point nearer than infinity, so as to bring both maculæ to bear on the same point. Obviously this power will depend not only on the condition of the converging muscles, but also on that of their antagonists.

The converging muscles (internal recti) act co-ordinately with the ciliary muscles in looking at near objects. Convergence and accommodation are thus intimately associated. At the same time it can be shown experimentally (by paralysing the accommodation with atropine) that their action is independent, and the amplitude of the former is, as a matter of fact, somewhat greater than that of the latter.

LENSES.

The ordinary case of lenses employed in vision testing and the estimation of errors of refraction contains convex, concave, and cylindrical lenses. These lenses are spherical, and are numbered according to their

power, the metrical system of numbering being now universally adopted. A lens of 1 metre focus is taken as a unit and called a diopter (1 D); a lens of 2 D has a focal length of half this, 50 c.m.; that of 4 D has a focal

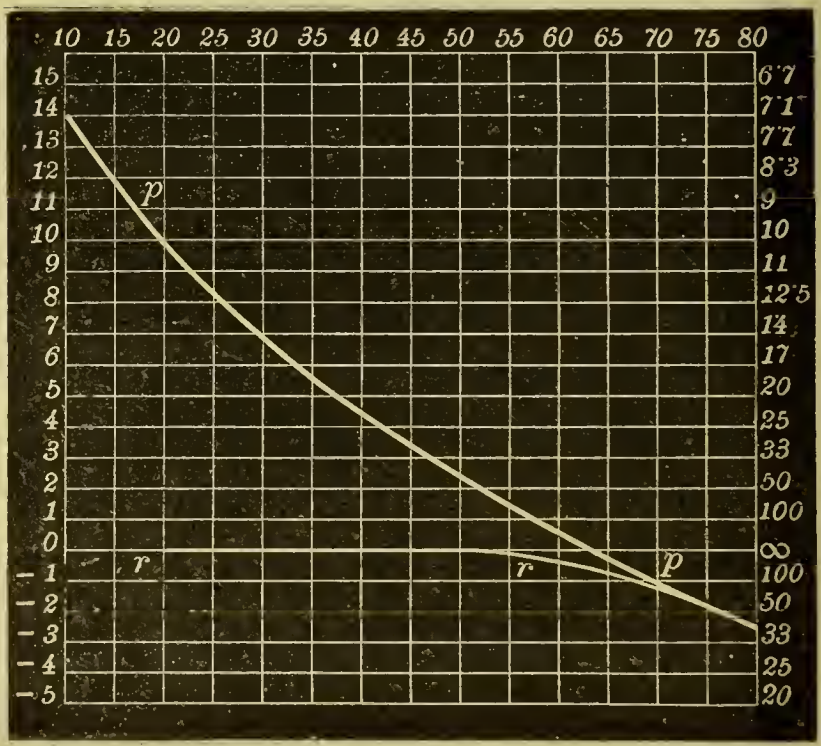


FIG. 4.—RANGE OF ACCOMMODATION AT DIFFERENT AGES. (AFTER DONDERS.)

length of 25 c.m. Lenses between the diopters are shown by decimals, thus: ·50 D, 1·25 D, 1·75 D, etc. Concave lenses are known as *minus* lenses, convex as *plus*—the former diverging, the latter converging rays impinging on them.

Cylindrical Lenses.—If we imagine a round pole or stick made of glass, a glass cylinder in fact, we can easily see that rays of light passing along its

axis will not be deflected, while rays perpendicular to the axis will be affected by the convex surface. Cut the cylinder through lengthwise and you convert it into two

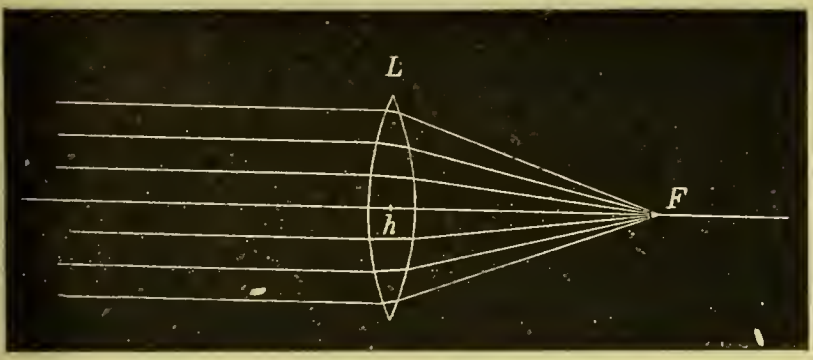


FIG. 5.—UNION OF PARALLEL RAYS, EFFECTED BY A CONVEX LENS. (FUCHS.)

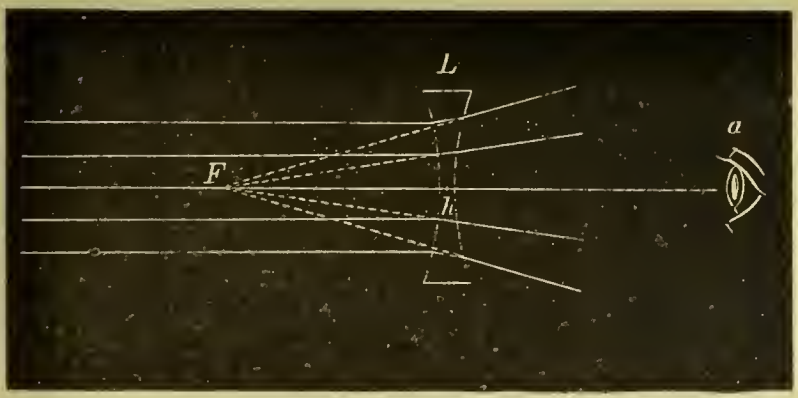


FIG. 6.—DISPERSION OF PARALLEL RAYS BY A CONCAVE LENS. (FUCHS.)

cylindrical lenses—lenses plane in one meridian, convex in the other; what are called + cylindrical lenses. A − cylindrical lens, on the contrary, has its curved meridian concave. In the cylindrical lenses in the case the position of the axis is sometimes indicated by short

lines on opposite sides of the plane meridian, sometimes by "buffing" the glass for a short distance on opposite sides of the curved meridian. The minus and plus cylinders are numbered in the same way as the other lenses, but do not, as a rule, go beyond 5 D.

Lens cases also contain a spectacle frame, known as a "trial frame," black discs for obscuring one eye while examining the other (an envelope or other piece of paper stuck behind the trial frame is better), and very often prisms and red and green plane glasses. The use of the latter will be seen as we go on.

To determine the refraction of a given lens—say you want to know what the lenses in a pair of spectacles are—hold the lens in front of your own eye, look at the types or any other object through it, and then move it from side to side and up and down. If the object seen moves *against* the lens movement, the lens is convex ; if *with*, concave ; if it does not move at all, you are looking through a plane glass. To find the exact refraction place lenses of the contrary sign in front of it successively till you find one which neutralises it ; this neutralising glass gives the number of the lens. Thus a lens is found to be + because the type moves against it ; successive − lenses are applied to the front of it till the type becomes stationary. The − lens that produces this appearance is a − 3 ; + 3 therefore is the lens tested. A little more trouble is necessary in working out astigmatic lenses, especially if the meridians be oblique, but with a little patience it is easily done.

THE SENSE OF SIGHT.

The sense of sight is threefold, including Form Sense (the power of distinguishing the form of objects—Visual Acuity), Colour Sense (the power of distinguishing colours), and Light Sense (power of distinguishing variations in the intensity of light). In the next chapter I propose describing the methods adopted in testing these sub-senses, giving illustrations of the notation and abbreviations adopted in practice. In Army practice the tests for visual acuity by means of "Test Types" and the "Test Dot Card" are by far the most important, next in importance being the colour test by means of "Holmgren's Wools."

CHAPTER II.

VISION TESTING.

It is evident that the smaller the object seen, or the greater the distance at which an object is visible, the greater is the visual acuity. The visual angle (*vide supra*) subtended by the extremities of the object at the nodal point of the eye, furnishes a convenient means of standardising visual acuity, and is made the foundation of a system of testing the form sense which is both scientifically and practically satisfactory. What we have to do is to find the smallest image whose form can be distinguished by the normal eye; that is, the image whose terminal points subtend the least visual angle compatible with clear vision. An obvious mode of testing would be to carry an object farther and farther away from the eye until the farthest point at which it is visible is reached. This, however, presented obvious disadvantages, and so the more practical method of placing objects of different sizes at a constant distance was evolved.

A single dot is unsuitable as a test object, inasmuch as its visibility depends on its luminosity rather than on its size : *e.g.* the stars are brilliant points to the naked eye, yet are still only points when seen through the most powerful telescope. It is easy, however, to determine the greatest distance at which *two* dots are visible as separate objects, and from this to calculate the minimum visual angle. This minimum visual angle has been found experimentally (by Volkmann) to be 1°.

Test Types.—Snellen's Test Types, the authorised Army test for visual acuity, are founded on this fact. In these types letters of the alphabet (or, to meet the case of illiterates, various figures such as squares, circles, crosses, etc.) are arranged in rows, each row containing

letters of the same size, with a number over it showing the distance at which the letters in the row should be seen by the normal eye. Each letter is within a square, the sides of which are divided by lines into five parts; each part subtends the minimum visual angle of 1°, and consequently the whole letter subtends an angle of 5° (see Fig. 7). The top row should be seen at 60 metres, the next six rows at 36, 24, 18, 12, 9, and 6 metres respectively, from above downwards. The system on which letters are arranged so as to subtend the same visual angle of 5° by varying their size according to the distance will be divined at once by glancing at the diagram (Fig. 8).

FIG. 7.—A LETTER FROM SNELLEN'S TEST-CARD.

In carrying out the test the letters are placed in a good light (preferably artificial, as this is not subject to variation) at 6 metres, or roughly 6 paces, from the patient. Should the room be too small to permit of this, types (which for this purpose can be obtained

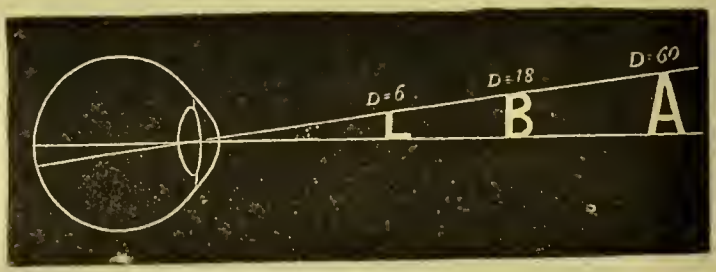


FIG. 8. (HARTRIDGE.)

reversed) may be reflected in a mirror facing which the patient stands. Obviously, then, the eye which reads the 6 metre type at 6 metres has normal vision. For the sake of brevity and convenience visual acuity is expressed by a fraction, the numerator of which shows the distance at which the patient stands, the denominator the distance at which the row of type seen should be read. With normal visual acuity, then, both numerator

and denominator will be 6, and the visual acuity of an individual with normal vision in both eyes would be thus noted :—

$$\text{Visual acuity (or V.A.)} = \text{R}\,\frac{6}{6}\,\text{L}\,\frac{6}{6}$$

Now take a patient who can only see the 24 metre type with one eye (right), and the 36 with the other (left), we would express his acuity thus :—

$$\text{V.A.} = \text{R}\,\frac{6}{24}\,\text{L}\,\frac{6}{36}$$

Perhaps we find the patient cannot read even the 60 metre type at 6 metres ; he must then be brought nearer and nearer until he sees it at (say) 2 metres with his right, 1 with his left, eye. Express this vision as :—

$$\text{R}\,\frac{2}{60}\,\text{L}\,\frac{1}{60}$$

Should he be unable to see the top letter at 1 metre, try if he can count figures, detect hand movement, or distinguish light from darkness. The following are examples of the mode of noting results :—

R—Counts figures at $\frac{1}{2}$ metre.
L—Hand movement.

R—P.L. (perception of light).
L—Nil.

So much for distant vision ; the distance at which the patient stands (6 metres) practically excludes accommodation.

In testing near vision we employ another set of Snellen's types, made up of small print of different sizes. We have to find out with them the smallest type read by the patient, and the greatest and least distance at which he can read it. The smallest type is known as ·5 (*i.e.* should be read at 50 c.m. or $\frac{1}{2}$ metre), next come ·6, ·8, 1, and so on to the largest, 4. In practice it is usually only necessary to put down one distance, viz. that at which the patient prefers to read it.

The near and distant vision of an emmetrope would, then, be shortly expressed thus :—

R $\frac{6}{6}$. Snellen ·5 at 50 c.m.

L do. do.

(In a young person with $\frac{6}{6}$ it is, however, quite unnecessary to measure the distance at which the ·5 is read.) In an ametrope, the record might perhaps be :—

$$R\,\frac{6}{12}.\quad \text{Sn. 1 at 20 c.m.}$$

$$L\,\frac{6}{24}.\quad \text{Sn. 1·75 at 15 c.m.}$$

Personally I prefer Jäger's test types for near vision, probably because I have been in the habit of using them for many years. In them the letters are of the ordinary shape and of the various sizes used by printers ; but they are not arranged on a scientific basis as Snellen's are. In the latter each letter, when held at the prescribed distance, subtends a visual angle of 5°.

The examination by test types should not end without trying whether + or − lenses placed in front of the eye improve vision. For instance, a − 2 D lens placed in front of a $\frac{6}{24}$ eye may convert it into a $\frac{6}{6}$ eye, thus recorded :—

$$\frac{6}{24} - 2 = \frac{6}{6}$$

If the eye be not improved by + or − spheres, note the fact thus :—

$$\frac{6}{24} \text{ not imp. by sph.}$$

Even if the eye sees $\frac{6}{6}$, you should try whether $\frac{6}{6}$ can *still* be seen (do not ask if it can be seen *better*) with a + lens in front : this will show, as will be seen later on, whether there is manifest hypermetropia or not. The highest + lens through which the patient can still see $\frac{6}{6}$, known as the highest + lens he will "accept," is the measure of this "manifest hypermetropia" or

"Hm." An eye seeing $\frac{6}{6}$ both with and without a + ·75 lens is thus noted :—

$$\frac{6}{6} + \cdot 75 = \frac{6}{6}$$

or as some oculists put it :—

$$\frac{6}{6} \text{ Hm. } \cdot 75$$

Should a + lens dim the vision of $\frac{6}{6}$ you can put :—

$$\frac{6}{6} \text{ no Hm.}$$

The near point in emmetropes recedes after the age of forty-five beyond 22 c.m. (*vide supra*), earlier in hypermetropes, later in myopes. In an emmetrope of forty-eight, then, we will probably get the following result :—

$$\frac{6}{6} \text{ Sn. } 1{\cdot}25 + 1{\cdot}5 = \text{Sn. } {\cdot}5$$

On trying an astigmatic patient with the test types his V. will always be found below normal ; he will read some letters with ease, others with difficulty or not at all ; and, if his meridians are oblique, he is very apt to put his head on one side, possibly with a view to bringing his own meridians and those of the types parallel (Hartridge). Snellen's "sun-burst," Pray's striped letters, and a movable hand on a clock face, have been used to detect and estimate astigmatism ; the patient will see certain rays in the "sun-burst," certain striped letters, the hand of the clock in certain positions, more distinctly or less distinctly as the case may be. Now that retinoscopy is so universally employed we do not waste time over these tests, the execution of which is tedious and the result often fallacious, but simply note the vision as "not improved by spheres," and proceed to determine the exact refraction by retinoscopy or, if we are very experienced ophthalmoscopists, by "direct" examination.

Test Dot Card.—Every Army surgeon is familiar with the "Test Dot Card" (Army Form I. 1,220) em-

ployed in testing the vision of recruits. This is a card about the size and shape of an ordinary playing-card, on which black circular discs, $\frac{1}{5}$ inch in diameter, are printed at irregular distances from each other, but with no interval less than $\frac{1}{5}$ inch. A plain card is arranged to slip over this, and so vary the number of dots the recruit is asked to count. When held at 10

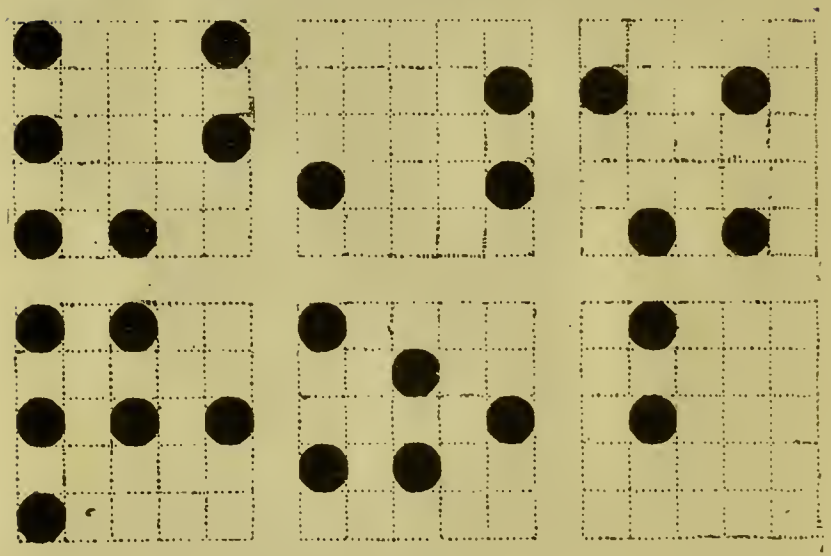


Fig. 9.

feet from the observer these $\frac{1}{5}$ inch dots are presumed to form retinal images equal to a bull's-eye 3 feet in diameter on a target 600 yards away; thus:—

600 yards : 3 feet :: 10 feet : $\frac{1}{5}$ inch.

Now it has been shown that the normal eye can count these dots at 43 feet; hence (10 : 43 :: 1 : 4·3) a recruit for the regular Army (who stands at 10 feet) may pass with one-fourth normal vision, and one for departmental corps, Militia, and Volunteers (who stands at 5 feet) with only one-eighth. Round dots of a certain diameter require a larger visual angle than parallel lines of equal diameter.

These dots should only be employed as a quick method of determining whether a recruit comes up to this minimum visual standard. If he cannot count them he should be dismissed at once, but it by no means follows that he should be accepted because he can count them. He may count them accurately and quickly with each eye separately, and yet have grave errors of refraction or graver disease which should cause his ultimate rejection. The dot test should be only the first of a series embracing types, focal illumination, and the ophthalmoscope. I may here mention, as illustrating the danger of relying on the dot test alone, three cases which I have seen recently. Case 1: A boy of nineteen with hypermetropia of + 8 in one eye, + 7 in the other. Case 2: A girl of twenty-two with advanced retinitis pigmentosa and pin-point fields of vision. Case 3: A bad case of quinine amaurosis with pin-point fields. All three counted the dots accurately; yet No. 1 suffered from constant severe headache, while 2 and 3 had the greatest difficulty in moving about alone owing to their "telescopic" vision.

A recruit who counts the dots but is found to be myopic should not, *cæteris paribus*, be rejected. Myopes of 1·5 and even 1·75 D will often count them easily.

The time is bound to come when the English, like the foreign, soldier will be permitted to wear glasses, and then comparatively high errors of refraction, if unaccompanied by disease, can be passed.

Testing Colour Vision.—The Colour Sense is the ability to distinguish colours, or, to put it more scientifically, lights of different wave-lengths. The usually accepted theory of colour perception (only a theory, however) is that known as the Young-Helmholtz, which assumes that three "fundamental" colours—red, green, and violet—produce three different sensations in the retina, the sensations produced by other colours being a mixture of these. Colour blindness is comparatively common, 3·5 per cent. of men and a little under 1 per cent. of women being colour blind; it was first accurately described by Dalton, himself colour blind. It may be congenital or due to disease. Total colour blindness is rare; partial, *i.e.* inability to distinguish

certain groups of colours, being the usual form. Red blindness and green blindness almost invariably go together. Violet blindness is extremely rare.

According to the Young-Helmholtz theory all colours act on the three sets of colour-perceiving retinal elements, but in varying degrees. Red acts strongly on the red-perceiving elements, feebly on the others; green strongly on the green elements, feebly on the others, and so on. Now let us assume a case of absolute *red* blindness only. Here red rays have no red retinal elements whatever to act on, but act feebly on the green and violet elements; while green and violet rays act strongly on their own perceiving elements. Hence red rays are seen as "something dark" compared with the brilliant green and violet rays; and the red-blind man, though totally unable to *see* red, does see that there is something in its place which is *not* green or violet. He may never, perhaps, know that his colour perception is different from the normal, for as he grows up he learns to call "red" the "something dark" which he sees; generally, too, he has a marked sensitiveness to variations in brilliancy which, of course, assists in disguising his defect from himself and others. Professor Fuchs tells a story of a physician who, having been asked to test the colour vision of certain railway employees, came to him to learn the technique of the test; much to his surprise and disgust, he soon showed that he himself was colour blind.

In the supposed case of red blindness we have been discussing the different colours are presumed to have been equally illumined; the red-blind man then distinguishes red by the fact that it looks darker than the others. If, however, the green and violet colours be gradually and carefully "toned down" to a point at which they only give the red-blind man the same sensation he gets from red, it is obvious he can no longer distinguish them from red. Such toned-down colours are called "confusion colours," and are important in testing colour vision by "Holmgren's Wools"; they are best produced by a colour-blind painter toning down different colours till they appear the same to him.

Holmgren's Wools are simply a number of different

coloured skeins of wool. In testing with them we do not ask the person examined to *name* the colours of the skeins, partly because, though not colour blind, he may be so ignorant as to be unable to name correctly the colours he sees, and partly because he may be colour blind and anxious to deceive the examiners—and this, with his marked susceptibility to differences of illumination, and perhaps previous practice with the wools, he may succeed in doing if asked to name the colours. We therefore give him a sample of a particular colour, and simply ask him to place beside it all skeins that look like it. We begin with a "pure" pale green skein, because if he matches this correctly his colour vision is normal, and we need go no further. Should he, however, place beside the green skein the "confusion-colour" skeins (grey, buff, etc.) then he is colour blind.

We do not, as a rule, except for scientific purposes, require to go beyond this to discover what is the *kind* of colour blindness. If we do, a pink (*i.e.* blue and red mixed) skein is placed before him. If he selects a blue skein as a match for this, he evidently only sees the blue in the blue and red mixture, and is therefore red blind —and almost certainly green blind too (*vide supra*); if he also chooses a green skein as a match, he is so certainly. If he matches the pink skein correctly (after failing in the green-skein test), his colour blindness is incomplete. In violet blindness, which is so rare, orange, red, and purple skeins will be chosen as matches to the pink one.

In tobacco amblyopia a "scotoma" for red and green is very common; in other words, a particular patch (central) of the retina cannot distinguish red and green, while the peripheral retina can. It is discovered by using coloured patches with a perimeter (*vide infra*). In consequence of this scotoma, a man suffering from tobacco amblyopia will often think his friends look pale, as he is unable to see the red in their complexions.

Light Sense Tests.—Except for scientific purposes, it is rarely necessary to endeavour to estimate the exact degree of light sense. At Moorfields we used to test the light sense roughly but sufficiently by placing the tested person side by side with one of known normal

light sense (generally the examiner) opposite a row of test types, the illumination of which was gradually lessened until a point was reached when the patient (presuming he was deficient in the sense) could no longer read, while the examiner could. I have often used types similar to Snellen's, but with grey letters, for the same purpose. Where rigidly accurate work is required Förster's "photometer" is useful. This is a box, blackened on the inside; on its posterior wall is a plate on which black lines on a white ground are placed. Through a window in the box a light of 1 candle power is thrown on to the test lines—this window can be altered in size by means of a screw. The patient looks at the stripes through two holes in the front of the box. The box should be placed in an absolutely dark room. The patient is kept in the same dark room for a few minutes till his retina has adapted itself; he is then made to look into the box with the light window closed, the latter is slowly opened, and the degree of light sense estimated by the size of the window required in order to enable him to see the stripes on the plate.

Field of Vision.—The "field of vision" is the whole area seen by one eye when looking at a fixed point; the object fixed is seen distinctly by the macula, others less distinctly by the rest of the retina. The binocular field is the area seen by both eyes fixed on an object; it is rarely taken. The normal monocular field for white extends upwards about 50°, inwards a little less, downwards over 60°, outwards about 100°. For coloured objects the field is less (see chart, Fig. 10).

An instrument called a "perimeter," which is simplicity itself, is employed in testing the fields; outline charts, on which the result can be registered, are also obtainable. McHardy's "self-registering" perimeter is the best, and should be part of the equipment of all large hospitals.

The extent of the field may be ascertained roughly by putting a small white mark on a wall, making the patient stand two feet away from it, close one eye, and fix the mark with the other; another white object is then moved towards the fixed one and marks made—to be subsequently joined—at the spots all round where the

moving object is first seen. A very rough estimate can be made with even less trouble by getting the patient to fix the observer's nose, while the latter moves his fingers towards it from various points.

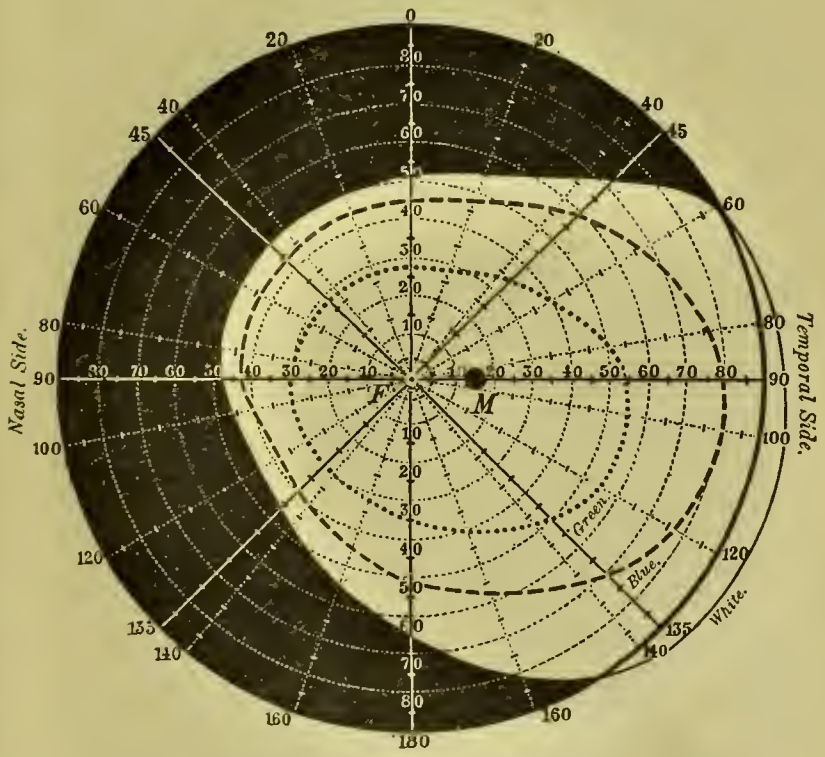


FIG. 10.—FIELD OF VISION OF THE RIGHT EYE FOR WHITE, BLUE, AND GREEN. (AFTER LANDOLT.)
F, Point of fixation: *M*, Mariotte's blind spot.

"Marriotte's blind spot" is the normal gap in the visual field (a natural scotoma) about 15° external to the point of fixation (see chart), corresponding to the entrance of the optic nerve. Schoolboys have a trick of detecting this spot by fixing the eye on one thumb and

then moving the other thumb slowly away from it; at a certain point the moving thumb disappears, to reappear as it moves farther on.

Eccentric vision is of great importance, as by its means we are able to guide ourselves in moving about. In certain diseases (*e.g.* pigmentary retinitis) the field may be reduced to the size of (on the chart) a three-penny bit. Such a patient may still be able to read $\frac{6}{6}$ slowly, "picking out" each letter as it were; yet his every movement will be attended with danger (*vide supra*—test dot card) as he only sees one small point in front, nothing at the sides, above, or below. Vision like this is known as "telescopic vision" from the obvious analogy. In tobacco amblyopia there is often a central scotoma for green and red (*vide supra*). Testing the field of vision is of great importance in certain diseased conditions of the eye, notably glaucoma.

Tension.—The tension of the eye should be tested before proceeding to examine it with the ophthalmoscope, and invariably before instilling a mydriatic for any purpose. Instruments for estimating tension have been invented, but I know of none better than the "tactus eruditus" of the surgeon, acquired by practice.

The eye is palpated through the closed lids by two fingers, just as fluctuation is tested for, and the result compared with that obtained in the other eye, assuming the latter's tension to be normal. Tension is noted by means of the following expressions: "Tn" means normal tension; T + 1, T + 2, T +3, varying degrees of hypertony (increased tension); T − 1, T − 2, T − 3, degrees of diminished tension (hypotony). The eyes of the old are usually harder than those of the young.

CHAPTER III.

THE OPHTHALMOSCOPE.

Focal Illumination.—The next step in the systematic inspection of the eye and its adnexa, presuming that the lids have been inverted and the conjunctiva both of the tarsi and eyeball looked at, is the examination by focal illumination. Focal illumination simply means the focussing of light on the cornea by means of a strong convex lens such as is found in ophthalmoscope cases. Its *modus operandi* is as follows. The patient is placed behind and beside a light—candle, lamp, gas, or electric globe—in a "dark room"; then, by the aid of the convex lens, this light is brought to a focus on the cornea. The lens may even be used at a window by ordinary daylight, but the fact that it may be converted into a "burning lens" by strong sunlight should not be forgotten.

The expression "oblique illumination," so generally employed for this test, is an unhappy one, and leads a tyro to manœuvre in every way but the right one with his lens. The illumination may be oblique if wished, but the point to remember is that the lens must be held *in the path* of the rays proceeding from the light to the eye, not behind or in front of them.

By this means the cornea, iris, pupil, and front of lens can be examined and their condition noted. A milky appearance of the lens in the pupillary area should not hastily be assumed at this stage to be cataract. The pupillary reaction to light can be tested by flicking the illumination on and off the pupil, and the reaction of the two pupils compared. It is as well to mention here that the pupils should not be pronounced unequal without first carefully ascertaining that the light (day or other) falls on both equally.

Transmitted Light and Retinoscopy.—We now place the patient, with the light behind and beside or behind and above him. Standing or sitting about 120 c.m. from him, we then throw the light into the pupil by means of a concave mirror of 25 c.m. focus, and at the same time look at the pupil through the sight-hole of the mirror.

(The mirror forms part of most ophthalmoscopes, but the writer finds it much more convenient to have it entirely separate. A very convenient little case containing this mirror and the lens for focal illumination is made by Messrs. Curry and Paxton, of Great Portland Street; this case also serves for indirect ophthalmoscopic work.)

The pupil will now appear as a vivid red circular disc, the "fundus reflex," unless there be opacities in the media or a large retinal detachment; we are assuming that no opacities in the cornea have been detected by focal illumination. Opacities of the lens or vitreous will appear as black spots or lines on a red ground; if of the vitreous they probably float about. Occasionally we find no red reflex at all, owing to a cataract, or large vitreous opacity, or completely detached retina.

Should the optic disc or retinal vessels be seen in the illuminated pupil, the patient must be myopic or hypermetropic. The optical explanation of this and of the shadow test is though simple a lengthy one; hence I do not propose to enter into it here.

We now proceed to rotate the mirror slightly on its vertical axis, *i.e.* from side to side: as we do so we perceive a black or blackish *shadow* appear at the edge of the pupil, which will cross its whole area if the mirror be rotated still more. The appearance and behaviour of this shadow form the basis of the "shadow test" for estimating refraction, first discovered by Cuignet, and generally known in this country as "retinoscopy." It is, on the whole, the best and most generally adopted method for the determination of refraction; but, simple as it is, it requires a little practice in order to cultivate a new faculty of observation.

If now we find that this shadow moves *with* the

mirror, the eye, or at all events its *horizontal* meridian (for we are moving the mirror from side to side), is myopic; if against, hypermetropic. Similarly, by tilting the mirror on its *horizontal* axis (up and down) we ascertain whether the *vertical* meridian is myopic or hypermetropic. Possibly we find the shadows move *obliquely* across: this is common enough in astigmatism.

The patient, unless atropised, should not look *into* the mirror, as if he does the pupil shuts up to, perhaps, pin-point size; but gaze into distance in a direction over the examiner's shoulder. Even then the pupil is often inconveniently small: for this and other reasons it is usual, unless the age of the patient or disease negatives it, to instil a mydriatic (atropine or homatropine) into the eye before examining it.

The rate of movement of the shadow is in inverse proportion to the amount of error of refraction: if the latter be high the former is slow, and *vice versâ*. A peculiar *glassy* grey appearance of the illuminated pupil, with the disc and vessels distinct, is very characteristic of high hypermetropia.

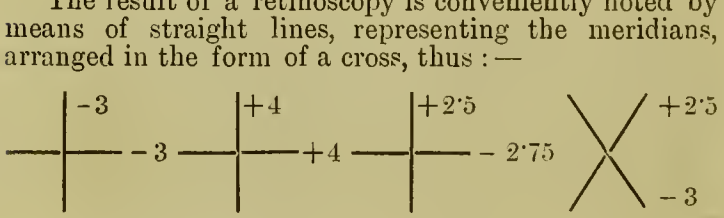
Now as regards the practical application of retinoscopy to the estimation of errors of refraction.

Place the adaptable trial-frame from your lens case on the patient's nose, and cover the eye not under examination with a black disc or piece of paper thrust behind the frame. Then, standing or sitting at a distance of 120 c.m. (about 4 feet) from him, apply the back of the mirror to your eye, and, looking through the sight-hole, throw the reflected light into his pupil, at the same time directing the patient to look at the wall over your shoulder if he be not under a mydriatic, or to look at the hole in the mirror if he be. If the shadow be *with* your mirror, drop successively stronger *concave* lenses into the trial-frame, until you reach a concave glass through which no distinct shadow, with or against, is seen. The degree of his myopia is then approximately 1 D *more* than this—*e.g.* if the glass be −3 D, his myopia is about −4.

If the shadow move *against*, try successive *convex* lenses. If a lens under +1 neutralises the shadow,

there is very low myopia; if + 1 neutralises, emmetropia. If more than + 1 be required, the case is one of hypermetropia, and we proceed to drop + lenses into the frame until one is reached which neutralises; the degree of his hypermetropia is then 1 D *less* than this—*e.g.* if the neutralising lens be + 5 D, the H. is only + 4 D.

The result of a retinoscopy is conveniently noted by means of straight lines, representing the meridians, arranged in the form of a cross, thus: —



Should the shadow move against in one meridian and with in another, or the same in both but requiring differing neutralising glasses, the case is one of astigmatism, and will require cylinders, or combined spheres and cylinders, to correct it.

Always remember, too, that the shadows may move *obliquely*, indicating oblique meridians.

Many ophthalmic surgeons employ a *plane* mirror for retinoscopy, and this method has some advantages. The observer stands 4·5 metres away from the patient; the circle of light thrown on the eye is much larger and less brilliant. The shadow movement is just the reverse of that seen with the concave mirror, viz. *with* in H., *against* in M., and no additions or subtractions are required. On the whole, however, the concave mirror will be found more convenient to work with; with a little practice the additions and subtractions are performed automatically.

In any case the results obtained by retinoscopy have often to be considerably modified in finally fitting the patient with glasses, as will be seen.

Indirect Ophthalmoscopic Examination. —This is also known as the examination of the *inverted image*. It is carried out with the concave mirror used in retinoscopy and the lens employed for focal illumination.

The patient is best placed with the light at his *left* side and behind the head; in this way both eyes can be examined quickly one after the other without moving the light. (If the light be on the right side, the natural curve of the surgeon's left arm will obstruct the path of the rays from the light to the mirror.) Now take the mirror in the right hand, apply it to the right eye, and throw the light into the right pupil as in retinoscopy, at the same time asking the patient to look at your right little finger extended for the purpose. On obtaining the red reflex, interpose the convex lens, held between left thumb and forefinger and steadied by the little finger on

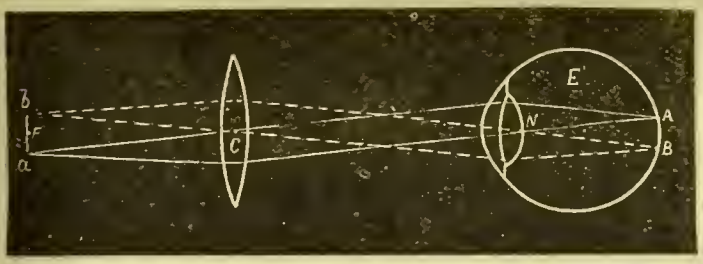


FIG. 11. (HARTRIDGE.)

the forehead, in the path of the rays from the mirror to the eye, about 7 c.m. (nearly 3 inches) from the latter. Now look at the *lens*, and you will see, *after some practice*, an *inverted* image of the fundus, magnified to about four times the natural size.

The same procedure is adopted for the left eye, with the exception that the patient should be asked to look at your *left ear*. The object of directing the patient's gaze at the little finger in one case and at the ear in the other is to bring the optic disc into view first.

As the optical explanation of the formation of this inverted image is short as well as simple, it is as well to give it here (Fig. 11).

The rays A B, projected by the mirror into the fundus, leave it again in pencils through the refracting media, emerge parallel, and are converged by the lens C,

which reunites them at A B in front of the lens, there forming the inverted image seen by the observer.

By a slight motion of the surgeon's head in one direction or of the lens in another, and later by getting the patient to look up, down, in, and out, the entire fundus may be examined.

Practice, often of considerable duration, is required to become expert in the indirect method; many, including the writer, have found the indirect more difficult to learn than the direct. It is valuable, inasmuch as by its aid one is enabled to obtain quickly a general view of the fundus. For the examination of minute details, however, the *direct* method is necessary; with it we not only see the various parts of the fundus in their proper relative positions (erect image), but we also see them magnified about 15 diameters.

Direct Ophthalmoscopic Examination.—Most ophthalmoscopes possess two mirrors, a large and small, both concave. The *small* mirror, which is tilted forward at one side so as to assume a wedge shape, is the one used in direct observation; the large one is the one employed in retinoscopy and indirect examination.

If the observer be inexperienced, the patient should be under the influence of a mydriatic so as to suspend his accommodation and dilate the pupil; an experienced ophthalmologist may be able to dispense with this.

The following is the *modus operandi*. As the observer has to look through the mirror, see that its sight-hole corresponds with that of the frame of the ophthalmoscope. Place the light behind and a little to one side of the patient's right ear if the right eye is to be examined, of the left if the left; use your right eye to examine his right, your left to examine his left. *To examine the right eye*, grasp the ophthalmoscope in your right hand, applying your forefinger to the cogged wheel by which the series of tiny lenses in the ophthalmoscope is passed across the sight-hole. These lenses are numbered in white figures for minus, red for plus. If you are emmetropic, turn the cogged wheel till 0 appears; if ametropic, turn on your correcting lens. If both you and the patient are emmetropic—and you should always begin with emmetropic patients—no lens will be required.

Turn the little mirror round so that the *apex* of the wedge is towards the right side of your nose; apply your right eye to the sight-hole at the back: throw the rays reflected from the mirror (taking care they are those reflected from the *small* mirror) into the observed pupil, and then bring the mirror with your eye still applied to it right up to the observed eye, as close to it as a spectacle glass would be. Do not "joggle" the ophthalmoscope about, but learn to keep the light steadily on the pupil; once you have learned to do this the battle is practically won. *In examining the left eye* the ophthalmoscope must be taken in the left hand—ambidexterity is essential to success in ophthalmic surgery, by the way—with the left forefinger on the cogged wheel, and turn the apex of the mirror wedge to the *left* side of your nose, applying the sight-hole to your left eye. Otherwise, follow the same directions as for the right, remembering, of course, that the light must be shifted to the patient's left side.

You will now see the red glow of the illuminated pupil. Endeavour to relax your own accommodation by looking at vacancy with your other eye (never close it). You are familiar with the fact that when reading a book you can relax your accommodation by looking *through* the book, the words then becoming indistinct. Try to look *through* the observed eye now in the same way. You will be rewarded by seeing the red blur become clearer; then you will see an obvious blood vessel. Trace this vessel to its origin, and then quite suddenly you will see the pretty, familiar picture of the optic disc with the vessels springing from it. The battle is now won; the rest is all a matter of practice. In a wonderfully short space of time you will find you can "get on to" the fundus at once, and study all its details by shifting your ophthalmoscope ever so little one way or another, getting the patient to look up and down, etc. As I said above, the battle is practically won when you can keep the red pupil *steadily* before the eye; and this, of course, like the development of any new muscular sense, requires training.

I admit that the learner will find all this more easily said than done, but he must not be discouraged

by preliminary failures; there is no royal road to the use of the ophthalmoscope; everyone must practise, practise, practise till proficient. Nothing but practice will enable the observer to throw the reflected light quickly into the observed pupil, *to keep it there*, and to suspend his own accommodation. In practical teaching I find it a useful plan to make the learner hold his ophthalmoscope close to the examined eye at once, until he is quite positive he is throwing the light from the *small* mirror into the patient's pupil and that he can keep it there; *then* let him, without moving the ophthalmoscope, bring his own eye up to the sight-hole

FIG. 12. (HARTRIDGE.)

at the back of the mirror. It is often advisable, too, to allow a young learner to put up a -1 or -2 lens at the sight-hole in looking at an emmetropic eye, as he cannot learn all at once to suspend his accommodation.

Having learned the trick of the thing, which, like swimming, once learned is never forgotten, proceed next to examine a series of normal eyes until you are thoroughly acquainted with the details of the normal fundus.

The accompanying diagram explains clearly the reason why the fundus of an observed emmetropic eye is seen clearly by an emmetropic observer.

The rays coming from the observed eye E at *a* issue parallel, and are focussed by the emmetropic observing eye E' at *b*, where a clear image of the observed fundus is formed. Should the observed eye be hypermetropic, the rays leave it divergent, and need a convex lens to focus them on the observer's retina; if myopic, they

leave convergent, and require a concave lens. It is self-evident, also, that the ametropic observer must correct his own error of refraction.

Having become thoroughly familiar with the normal fundus in the emmetrope, we now examine ametropic eyes. In *myopia* the details of the fundus appear indistinct, because they are focussed in front of your own retina ; to see the myopic fundus, then, you must turn on the minus lenses of the ophthalmoscope till you come to one through which the fundus is well seen. The weakest glass of this kind is the measure of the myopia, and this is a very good way of measuring it if you are sufficiently trained. One would naturally expect that, beginning with the lowest minus lens, the view of the fundus would get clearer and clearer through successively stronger lenses until finally it becomes quite clear through the correcting lens. This, however, is by no means always the case ; all ophthalmic surgeons are familiar with the more or less unexplained fact that lenses weaker than the correcting lens often make little or no difference in the view of the fundus, which yet will quite suddenly become clear seen through the actual correcting lens.

It is customary in books to say that in *hypermetropia* the fundus details also look indistinct, but become distinct when the plus glasses are turned on. This, however, is only the case when the surgeon has his own accommodation *absolutely* suspended. As a matter of fact, the fundus in hypermetropia nearly always looks beautifully *near* and clear, no matter how carefully the observer's accommodation be suspended. A little experience teaches the meaning of this near and clear appearance, and, presuming it to be due to hypermetropia, the surgeon turns on the convex lenses (red numbers) of the ophthalmoscope until he reaches the *strongest* one through which, with his own accommodation suspended, he can still see the fundus clearly. This strongest convex glass is, if the patient's accommodation be also suspended, the measure of the degree of H.

It is assumed that the observer looks at the *disc* in estimating in this way ; true, it is the macula whose

refraction we want to find, but the disc affords a much more sharply defined picture to examine, and, apart from that, light thrown direct on the macula is apt to cause the pupil to contract to pin-point size if the patient is not under a mydriatic.

In an astigmatic eye the disc, as seen through the astigmatic cornea, *appears* to be oblong, with its long

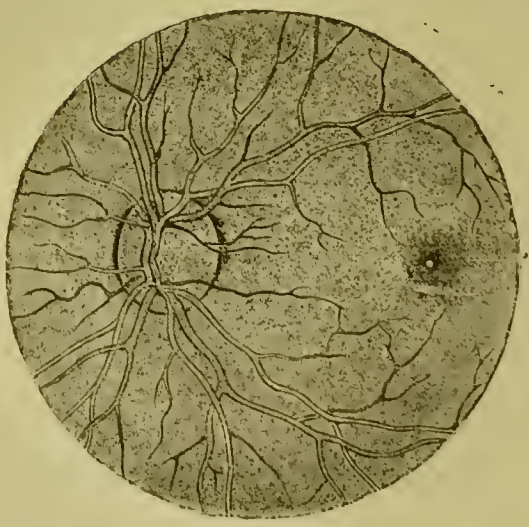


Fig. 13.—Normal Fundus of the Left Eye, seen in the Erect Image. (Fuchs.)

axis vertical, oblique, or even horizontal; the lens through which the vessels on one meridian of the disc are seen represents the refraction of the *opposite* meridian and *vice versâ* (see next chapter). The estimation of astigmatism in this manner is, however, practically impossible for the beginner.

The normal fundus is, or should be, a familiar picture to the student; it will be found portrayed in all atlases of ophthalmoscopy. Haab's Atlas (English edition) is small, cheap, and within its limits excellent.

The red colour of the fundus is produced mainly by

the chorioidal vessels, slightly reinforced by the retinal vessels, and possibly also by the "visual purple" contained in the retinal rods and cones (discovered by Bell). Dark persons have darker fundi than fair; in albinos the retinal and chorioidal pigments are absent, or at all events scanty, and the chorioidal vessels are beautifully seen as a red network on the white sclerotic.

The *optic disc* is seen as an oval greyish pink disc, with its long axis usually vertical; the nasal side is much pinker than the temporal. A physiological cup, or depression (discovered by the fact that lenses of different powers are required to see the floor and margin of the disc), at the temporal side is often present; it does not, however, reach to the edge of the disc, as does the *pathological* one seen in glaucoma. At the bottom of this cup will be seen the fine greyish lattice-work of the lamina cribrosa. Round the disc will usually be found a white ring, due to the fact that the chorioid does not quite reach the disc; round this again is a black ring, often very well marked, of chorioidal pigment: these are known as the *scleral* and *chorioidal rings* respectively.

A fairly common congenital anomaly, which should not be mistaken for a pathological process, is the existence of *medullated nerve fibres* in the retina. The normal retina is transparent because the optic nerve fibres lose their medullary substance before passing through the lamina cribrosa. Sometimes, however, the medullary sheath appears again in the eye, and gives rise to a bright white "frayed-out" patch extending from the disc into the retina, generally in an upward or downward direction. I have seen them, however, forming a brilliant flame-like ring all round the disc, and even situated at some distance from the disc with intervening normal retina. Medullated nerve fibres—often known as *opaque nerve fibres*—are normal in the rabbit's eye; they will be found beautifully pictured in Haab's little atlas.

Looking horizontally outwards from the disc (or inwards in the indirect method) we come to a more or less bright oval reflex from the retina, of a deep red colour. This indicates the *macula lutea;* in its centre is

a bright red spot, the *fovea*. The retina itself cannot be seen owing to its transparency, though it often sends out a shimmering reflection like watered silk (best seen in dark children).

The central vessels of the retina divide on the disc (sometimes before) into upper and lower branches, which almost immediately divide again, forming the superior and inferior nasal and temporal vessels. The small vessels supplying the macular region proceed from a delicate arch formed by the superior and inferior temporal vessels and by vessels from the disc; these minute vessels *appear* to end at the margin of the macula. The arteries differ from the veins in being narrower and lighter in colour, and in having a broader and lighter light-reflex. A *venous pulse* is often present at the disc, and may be produced in any eye by pressing the eyeball gently with the finger. It is a very pretty phenomenon; it looks as though a black shade were being pulled rhythmically backwards and forwards across a small dim lamp.

It occasionally happens that a small branch from the short posterior ciliary arteries (which form a ring round the optic nerve foramen) bends out from the optic nerve and enters the retina at the outer margin of the optic disc; thence it runs out to the macula. The existence of this vessel will often enable the victim of *embolism of the central artery* to retain fair central vision.

The inexperienced ophthalmoscopist will often mistake normal appearances, such as the physiological cup, a very black chorioidal ring, venous pulsation, "shot-silk" retina, slight blurrings of the disc-margin above and below, for abnormalities. Daily practice, with plenty of clinical material, however, soon makes the distinction clear. When anything unmistakably abnormal is detected, study it carefully, and learn before everything else to *describe* it accurately, conscientiously, and, if you can, picturesquely; never be precipitate in proclaiming a diagnosis.

Opacities in the media are easily studied by the direct method, by putting up a high + lens. Vitreous opacities are best seen through the little plane mirror,

first used, I believe, by Mr. Lang; many ophthalmoscopes are fitted with it, so arranged that by means of a hinge it can replace the small concave mirror.

Anyone who has read and worked so far can now make very useful "eye records" in his note-book; such records are always valuable, and it is well to note briefly every case examined. The form of such records will be best indicated by a couple of examples:—

1. Mr. A. B.—Vision $=\frac{6}{6}$ no Hm. Snellen's ·5 R. & L.
Colour perception perfect.
Fields normal.
Media clear.
Fundi normal.
Retinoscopy (concave mirror).
R. $-\Big|\frac{+\cdot75}{+\cdot75}$ L. do.

2. Mr. C. D.—Vision = R. $\frac{6}{60}-4=\frac{6}{6}$ Sn. ·5 at 10 c.m.
L. $\frac{3}{60}-7=\frac{6}{36}$ Sn. ·3 at 15 c.m.
Colour perception good.
Fields normal.
Media clear.
Right fundus — Crescentric atrophy of chorioid (myopic crescent) on right of disc.
Left fundus—Large myopic crescent. "Stippling" at macula.
Retinoscopy R. $-\Big|\frac{-3}{-3}$
L. $-\Big|\frac{-6}{-6}$ (concave mirror.)
Left pupil > right (*i.e.* larger than right).

Outline charts of the fundus, on detachable papers with gummed backs, can be obtained of most medical booksellers, and afford a convenient means of recording fundus changes. I have used those sold by Messrs. Bale, Sons and Danielsson for years, and find them most useful.

CHAPTER IV.

ERRORS OF REFRACTION.

Errors of refraction are very common, not only in civil life, but also in the Army, and familiarity with the means of diagnosing and treating them is a very essential part of the professional equipment of every Army surgeon. Of these, Hypermetropia is the most common, as an astonishingly large proportion of young adults suffer from it; Myopia is the most serious, as it is not only an error of refraction but a disease; Astigmatism is the most difficult to correct. Presbyopia is unusual in the rank and file of the Army, owing to the limits of age.

HYPERMETROPIA.

As Mr. Hartridge concisely puts it, hypermetropia is a condition in which the focal length of the refracting media is greater than the length of the eyeball. If we look back at Fig 2 B, we see that parallel rays falling on the hypermetropic eye are focussed *behind* the retina instead of on it, a "circle of diffusion" (causing a vague, ill-defined image) being formed on the retina, owing to the latter intercepting the cone of rays before it comes to a point. This condition may be due to shortness of the eyeball or low refracting power. The hypermetrope cannot see either distant or near objects clearly when his eye is at rest; for if he cannot focus parallel rays on his retina, he is still less able to focus divergent rays—and, after all, all rays are divergent, though rays from distant objects diverge so slightly that as a matter of convenience we call them parallel. If, however, rays from an object can by any means be made convergent, then the hypermetrope can focus them and see the object distinctly. In correcting hypermetropia, then, it is necessary to make parallel rays from distant objects

convergent. This can be done naturally by the exercise of accommodation, whereby the lens of the eye is made more spherical (or rather permitted to become more spherical); or artificially by interposing a convex (converging) lens in the path of the rays before they reach the eye.

All hypermetropes correct their refractive error to a greater or less extent by accommodating; but fatigue very soon results, for not only have they, like emmetropes, to accommodate for near objects, but also for distant ones. They are always using their accommodation in their waking moments, and their refraction is perpetually varying according to the state of freshness or fatigue of their accommodating apparatus; it varies not only from day to day, but from hour to hour, and often in the young to a very considerable extent. For example, a hypermetropic schoolboy may see clearly in the morning with + 1 lenses; in the afternoon, after his day's lessons, he may require + 2's to see as clearly; after instilling atropine into his eyes for a week, we perhaps find he requires + 5 lenses. This example also serves to explain the meaning of the expressions "manifest," "total," and "latent" hypermetropia. The + 1 or + 2 lens first required, when he is still using his accommodation, is the measure of his "manifest" hypermetropia; + 5 lens represents his "total"; and the difference between the manifest and the total, in other words the amount he naturally corrects, is the "latent" (here + 3 or + 4 D). Obviously, then, Nature cannot be trusted to correct hypermetropia, and artificial means, viz. spectacles with convex lenses, must be adopted.

Causes.—The commonest cause is a too forward position of the retina, which may be due to its protrusion by exudation or tumour, but is most usually owing to the fact that an otherwise normal eye is abnormally short—a congenital malformation. Another cause is diminished refractive power of the media, due to flattening of the cornea, absence of the lens (aphakia), or weakening of the power of the lens to assume a spherical shape. The latter form is seen in emmetropes of advanced age, who will be found to have hypermetropia as well as presbyopia.

Hypermetropia is by far the most frequent error of refraction. It is, I regret to say, very common, and a source of never-ending trouble in the Army, as a hypermetrope of even high degree has no difficulty in passing the "dot test" for recruits. A young hypermetrope can often read $\frac{6}{6}$ clearly, though perhaps only for the moment; and as the "dot test," at the most, only represents $\frac{6}{24}$ we can easily understand the frequency of hypermetropia amongst soldiers. Only a short time ago I examined a young soldier, and found he had H. + 8 in one eye, H. + 7 in the other; yet he could "count the dots" with ease at 10 feet. He "went sick" frequently with headaches, was a bad shot, and had often been unjustly suspected of malingering (see Chapter II.). The time will surely come when correcting glasses will be generally worn amongst all ranks in our Army; until it does, however, no recruit with total H. over + 4 should be accepted.

Symptoms.—Hypermetropic eyes are often noticeably small, even to the casual observer. Most newborn children are hypermetropic, but as they grow the eyes gradually elongate until they become emmetropic. Typical H. occurs when this elongation does not proceed to a sufficient degree, and the eye remains permanently too short. The hypermetrope, especially if young, may see distant objects clearly by using his accommodation; but for near objects the strain on the accommodation cannot be kept up long, and the objects soon become blurred and indistinct owing to circles of diffusion, instead of clear images, being formed on the retina. This blurring at first comes on only after prolonged near work, such as reading or writing; soon, however, it appears after a few minutes, and is apt to be accompanied by pains in the eyes and headaches. *Convergence*, which as we have seen, is intimately associated with accommodation, is also very liable to be affected; the eyes converge to a degree corresponding to the degree of accommodation, and as the strain becomes greater one eye fixes the object, while the other converges too much

squinting inwards (convergent strabismus), and this squint, at first only periodic, soon becomes permanent.

A hypermetrope is sometimes mistaken for a myope because he holds a book very close to his eyes; but many hypermetropes do this in order to get larger visual angles, and larger, though less clear, retinal images.

Presbyopia, as might naturally be expected, comes on earlier in hypermetropes than in emmetropes.

Estimation and Correction.—To show clearly the means we adopt for estimating and correcting hypermetropia, I shall give an example of the commonest type of case.

A boy of sixteen presents himself for treatment. He states that his eyes never troubled him till about a year ago, when he began working for an examination. He now complains of blurring of sight when he has worked an hour or so; he also gets pains in the eyes. Has had headaches, which he probably attributes to "biliousness," at recurring intervals almost as long as he can remember; latterly they have been very frequent. His candid friends tell him that he squints occasionally. Tried with the types he reads $\frac{6}{6}$ with each eye; with a + 1 lens each eye still reads $\frac{6}{6}$; with both together he can probably see $\frac{6}{6}$ with + 1·25 lenses (in other words "accepts" + 1·25). Vision is then at once recorded thus:—

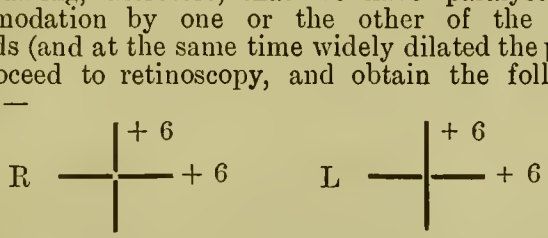
R. and L. $\frac{6}{6}$ Hm. + 1. Together Hm. + 1·25.

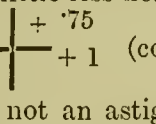
We have now estimated his manifest H. The best thing to do after this is to instil atropine twice or thrice daily for three or four days, or even a week, in order to thoroughly paralyse the accommodation and bring out all the latent H.; this, however, means that he will not be able to do near work for at least ten days, and he may not be able to afford the time. In that case the next best thing is to instil *homatropine* (the effects of which pass off within 24 hours) every quarter of an hour

for a couple of hours; if, during these two hours we make him wear, *and look through*, convex glasses much higher than the degree of his Hm.—say + 2·5's or + 3's —we can, as a rule, sufficiently paralyse his accommodation to be able to estimate the total H. pretty accurately.

Care should be taken in the employment of mydriatics, especially after the age of thirty-five; the possibility that they may induce glaucoma, or rather determine an attack in an eye predisposed to it, should never be forgotten. Many general practitioners prescribe atropine in nearly every eye-wash they order, with some vague idea that it is a panacea for all eye diseases; and all ophthalmic surgeons are familiar with cases in which terrible results have followed this senseless procedure. It will be sufficient here to say that mydriatics should never be instilled where there is any increase of tension in either eye; and only with the greatest caution after the age of thirty-five into any eye.

Assuming, therefore, that we have paralysed the accommodation by one or the other of the above methods (and at the same time widely dilated the pupil), we proceed to retinoscopy, and obtain the following result:—



It may be well to remark here that the refraction of the horizontal meridian is usually a little less normally than that of the vertical; thus 

(concave mirror) might be a perfectly normal, not an astigmatic eye.

His refraction, then, is + 6 in each eye with the concave mirror, so that the total H. is + 5 (*vide supra*).

An experienced ophthalmoscopist can avoid the trouble of retinoscopy by the employment of the *direct* method; here he would probably find the fundus clearly visible in each eye through a + 5; hence his "direct estimation" of the H would be + 5.

We now try our patient, still atropised, with the types again, and find he reads $\frac{6}{6}$ with + 4; he will hardly accept the full + 5. What glasses should now be ordered for constant use? It must be remembered that the ciliary muscle will never be so thoroughly relaxed naturally as it has been under atropine; hence we very seldom order the *full* correction, except in the case of very young children with low or medium H. in whom we are endeavouring to cure a strabismus by means of glasses. Patients vary greatly in the amount of correction they will bear, or "accept"; so that each case must to a certain extent be taken on its merits. Mr. Hartridge advises ordering hypermetropes glasses correcting the whole of the manifest and one-fourth the latent H.; but I think most ophthalmologists are in favour of correcting more than this. Mr. Treacher Collins, to whom I was chief assistant for several years, taught me to order two-thirds of the *total* H.; and both at Moorfields and since I have found this an excellent *general* rule. Following this rule this patient is ordered :—

+ 3·25 D Spheres R. and L. (Spectacles; constant use.)

The patient should be told he may not find these glasses comfortable just at first, owing to his habit of using up so much of his accommodation. When there is a good deal of latent H., as in this case, I very often order a morsel of atropine ointment to be put within each lower lid every other night for the first eight days he wears the glasses; this enables the patient to "live up to" his glasses. In the present instance the spectacles should be worn constantly; but when the total H. does not exceed + 2·5 or + 3 it is only necessary to wear glasses for near work; always excepting children with a squinting tendency, who should wear them always.

Ciliary Spasm.—In some cases, by no means infrequent, of H., the overwork of the accommodation induces *ciliary spasm*. In such a case myopia may be simulated, as the spasm of the ciliary muscle permits the lens to become so spherical that parallel rays are actually focussed *in front* of the retina, so that the

patient cannot read $\frac{6}{6}$ without *minus* glasses. I have seen more than one hypermetrope wearing concave glasses (which had been purchased in shops) for this reason. The existence of this class of cases should never be forgotten, and it is always well to suspect H. when there is a history of sudden onset of short sight in young people. Cases of ciliary spasm generally require at least a fortnight's thorough atropisation before estimating and correcting.

Aphakia.—A very high form of H. is the result of the absence of the lens from the pupillary area. *Aphakia*, as this is called, is usually the result of cataract extraction, but may be due to luxation of the lens from injury ; congenital aphakia being the result of luxation during intra-uterine life. In looking into the aphakia eye by the direct method the observer finds he can see the fundus clearly through a very high (+ 10 or + 12) lens ; the absence of the lens may, however, be made certain by holding a candle in front of the eye, when only one image of the flame—the corneal one—will be seen. Were the lens present two other images would be visible—one on the anterior, the other (inverted) on the posterior, surface of the lens. In aphakia all accommodation is lost. To correct it a high convex lens, about + 12 probably, will be required for distant vision ; and a still higher one, + 16, for near work. In aphakia the result of cataract extraction there is nearly always some astigmatism also to be corrected.

MYOPIA.

Myopia is the opposite error of refraction to hypermetropia ; but is much more serious, inasmuch as it partakes of the nature of a disease. In myopia parallel rays are focussed in front of the retina, cross, and form diffusion circles on it (Fig. 2, *c*). Myopes often screw their lids together in an attempt to make these diffusion circles smaller ; to this is due the name (μύειν, to blink ; ὤψ, the sight)—a foolish name to retain, but it serves. To a myope an object situated at what is

conventionally called infinity is invisible or very indistinct; but if the object be carried nearer and nearer to his eye it finally reaches a point at which the rays from it are so divergent that they are focussed on the retina and form a clear image there. This point is the *punctum remotum* of the myopic eye (see Chapter I.). The greater the M. the more divergent must rays from an object be to be focussed clearly; hence the greater the M. the nearer must the far point be.

The rays from an object at infinity cannot be focussed, but rays from the far point (R.) can, as they are sufficiently divergent. Now, if we could convert the parallel rays from infinity into rays diverging to the same extent as those from R., it is obvious we should correct the myopia; and this is what we do, or attempt to do, in the actual correction—that is, we place in the path of the rays to the eye such a concave glass as will make the rays diverge as they do from R. I use the word "attempt" advisedly, as, unlike hypermetropia, myopia is too often not only an error of refraction, but also a disease impairing, or even destroying, the sensitiveness of the retina.

Granting that a concave lens diverging rays from "infinity" to the same degree as rays from R. are diverged will correct the M., it will be obvious that a glass whose focal length is equal to the distance of R. from the eye will be the correcting lens; and conversely the glass with which infinity is seen distinctly gives the distance of the far point and the measure of the M. Say a patient sees remote objects clearly with a -4 lens, then his far point is the focal length of that lens, *i.e.* $\frac{100}{4}$ or 25 c.m., and the degree of his M. is 4. Remember, however, that he will also see clearly with, and *possibly prefer*, a -5 or -6 glass, for he can easily neutralise the extra 1 or 2 D by accommodating; hence in testing with the trial case we stop at the *weakest* concave glass with which distant objects are distinctly seen. But it is not safe to assume that concave lenses chosen in this way are the proper correcting glasses to order, for this mode of estimating is, after all, a sub-

jective one. To ensure correctness we must also examine the eye ophthalmoscopically (retinoscopy or direct, or both); this examination may confirm, modify, or *even reverse* (*vide supra*, Ciliary Spasm) the impression derived from the trial lenses.

Causes.—Myopia is most commonly the result of elongation of the eye, so that the retina lies *behind* the point at which parallel rays are focussed. This elongation is due to stretching of the sclera, the posterior part of which is almost invariably the part affected, so that it bulges out backwards next the optic disc, forming what is known as a posterior staphyloma. Myopia, however, is not always due to elongation of the eye; it may be caused by excessive refraction of the cornea or lens. Conical cornea (always with astigmatism), luxation of the lens within the pupillary area (as the tension of the zonula is gone), ciliary spasm (spurious M.), and increased density of the lens (as in commencing senile cataract) are examples of this refraction group of cause.

A man with *moderate* non-progressive M. is by no means badly off. True, his distant vision is indistinct, though probably sufficient for ordinary purposes; but, on the other hand, he has very useful vision for near work, he has to use less accommodation than an emmetrope, and presbyopia sets in later. A myope whose far point is 22 c.m. need never (theoretically, at all events) wear presbyopic glasses, as his far point always remains the same, and he will not want to see nearer. A glance at the table in Fig. 4 enables us easily to calculate when, if ever, a myope of a given degree will have to wear glasses for near work.

A *high* myope, however, is in very different case. He uses little or no accommodation for near work, as his far point is so near; and if he does not wear glasses he practically loses the power of accommodating, and with it a good deal of the power of converging. Owing, however, to the closeness of his far point, he must try to converge to see near work clearly with both eyes; the result of the attempt often is that one internal rectus tires, and that eye diverges out (divergent strabismus). It may be added that the shape of the eye in M. presents

another obstacle to convergence. Changes in the fundus, too, are only too likely to appear, producing loss of visual acuity; so that his vision, even through his (theoretically) correcting glasses, will be indistinct. The stretching of the coats of the eye in progressive M. produces important atrophic changes in the chorioid and retina, first next the disc, later in the macular region; the vitreous becomes more and more fluid, and floating opacities appear in it; the lens may become opaque; and that terrible and only too frequent complication, detachment of the retina, may supervene.

The so-called "myopic crescent" is a crescent-shaped whitish patch of atrophy of the chorioid (in which atrophy the retina participates to a greater or less extent), embracing the optic disc, most frequently at its outer side; brownish markings, due to chorioidal pigment, are generally seen on it, especially towards the edge farthest from the disc, while the disc itself may be so displaced as to appear of oblong shape. This atrophic patch occasionally assumes a triangular shape with the apex towards, or even reaching, the macula; sometimes it may form a ring encircling the disc. *Coloboma of the optic nerve* (a coloboma is a cleft caused by arrest of development) is occasionally mistaken for the myopic crescent; it takes the form of a crescent, is observed only at the lower margin of the disc, is congenital, and almost invariably associated with defective visual acuity. The myopic crescent is always found in M. over 5 D; in lesser degrees it is often absent. An experienced ophthalmologist can often—but by no means always—tell from the appearance of the crescent whether the M. is progressive or not; but it is impossible to lay down hard and fast rules for guidance.

Changes at the macula, from "stippling" or "dappling" to hæmorrhages and large atrophic patches, are of frequent occurrence in high M., and always of grave import. Vitreous and lens changes and retinal detachment need not be further alluded to here (*vide* chapter on Injuries).

Myopia, unlike hypermetropia, is rarely congenital.[1]

[1] De Vries (Report of Netherlands Ophthalmological Society, 1901, I., No. 6) examined the refraction of 78 newborn children,

It usually develops during the growing age in those who strain their eyes over much at near work; hence school children, students, seamstresses, printers, tailors, etc., are especially prone to it. *Race* appears to play some part, as undoubtedly the proportion of myopes to population is much greater in some countries than in others. The constant strain on the accommodation and convergence necessitated by near work distends the tunics of the eye, and they finally "give" at the posterior pole. Schools, factories, workshops, each and all contribute their quota to the vast aggregate of myopes.

Rules for the supervision of large institutions with a view to the prevention of myopia are beyond the scope of this work. Army medical officers, however, should always visit the schools, tailors' shops, etc., at their barrack inspections to see that they are properly illuminated, and that benches and tables are properly constructed. In schools the light should be so arranged that it falls on the scholars' work from the left side; seats and tables should be adapted to the varying heights of the children, so as to obviate over much bending forward; and fine print and fine fancy work should be banished.

Under present rules a myope of more than 2 D cannot enter our regular Army; with more than this he could not "count the dots" as a recruit, or read $\frac{6}{24}$ as a candidate for a commission (*vide* Regulations for Medical Services). Myopes of higher degree (but not more than 4D) can, however, pass the dot test for recruits for the R.A.M.C., departmental corps, Militia, and Volunteers. In other armies, more especially in the Austrian and German services, myopes of much higher degrees (up to 6·5 or even more) can be admitted, provided their visual acuity reaches a certain standard; correcting glasses being, of course, worn. I shall revert to this subject later. (Appendix.)

Before entering on the subject of estimation and correction of myopia, I cannot do better than call attention to the following propositions laid down by Mr.

and found myopia in 5, hypermetropia in 62, and emmetropia in 11.

Priestly Smith,[1] which furnish valuable indications for the prognosis and treatment of this disease.

1. **Age.**—The younger the patient when myopia develops the more likely it is to increase. The majority of cases come to a standstill between fifteen and twenty-five.

2. **Degree.**—The higher the M. the more likely it is to increase.

3. **State of Chorioid.**—The higher degrees are to be feared even less than the lower, if in the latter the chorioid and retina are affected.

4. **Constitutional Changes.**—These are of the utmost importance. M. frequently follows grave constitutional disturbance and diseases causing high arterial tension.

5. **Evidence of Heredity.**—This is sometimes very marked, and as a rule in these cases, although the degree may be high, yet fundus changes are not so frequently met with, and therefore with a strong family history the prognosis is, as a rule, better.

6. **Occupation.**—This is of great importance. Excessive close work develops or aggravates it, and a person developing the disease should never be put to work necessitating this, at least not until any tendency to increase has been checked.

Estimation and Correction.—An example of an ordinary uncomplicated case is the following:—

A young man of twenty-one presents himself complaining that he sees distant objects badly, and can only do near work by holding it very close. Vision $= \frac{6}{60}$ $- 4 = \frac{6}{6}$ and Sn. ·5 at 15 c.m. R. and L.

Retinoscopy: $\frac{-3.5}{-3.5}$ R. and L.

[1] Annual Meeting of British Medical Association, 1901, Ophthalmological Section.

Direct examination: A – 4 lens required in order to see fundi plainly. No M. crescents.
Ordered – 4 D sph. R. and L.; constant use.

The glasses ordered *fully* correct the M.; and, though the full correction may worry him a little at near work just at first, I believe it to be best in young moderate myopes to fully correct the M. both for near and distant vision. He should also be instructed to hold his work at a proper distance, to avoid stooping over his desk, and to arrange the latter so that the light falls on it from behind.

In high M. it will be found that the full correction will not be "accepted" for near work: in such cases Mr. Hartridge's rule is an excellent one. Subtract the glass (say a 3 D) whose focus represents the distance (here 33 c.m.) at which work has to be held from the full correction; the difference will then be the glasses to be ordered for near work, while for distant vision nearly the full correction may be ordered.

For cases of very high M.—say over 14 D—without serious fundus lesion, *needling* (discission) of the lens in order to procure its ultimate absorption is becoming more and more a matter of routine practice in carefully selected cases. Most excellent results are obtained in this way, more especially in cases where the M. is evidently progressive and already perhaps causing commencing fundus lesions; in such cases it seems to arrest the progress of the disease, and by improving nutrition increase the visual acuity. The belief long held that needling increased the liability to detachment of retina is now generally held to be groundless. Most surgeons follow up the needling by a "curette evacuation" at a later date: Mr. Swanzy, however, prefers simple needling, believing the subsequent slow absorption to be a safeguard against complications. The result of the removal of the lens is, of course, that the power of accommodating is lost. A curious fact (but easily explained optically) is that the loss of refractive

power in the eye is generally 16 or 18 D, much more than the loss of the refracting lens accounts for. Hence a myope of — 14 will probably require no glasses for distant vision after the operation ; while for near work he will need *convex* lenses.

Patients with very high M., with advanced and advancing fundus changes, cause the surgeon the greatest anxiety, especially as they are often highly intellectual people engaged in literary pursuits, to whom sight is everything. Such cases often require extended periods of absolute eye rest. This rest is best attained by stopping near work, ordering *plane* smoked glasses to be worn, and instilling atropine daily for some weeks ; after a few days, a very little reading (with convex glasses if needed) may be permitted. I do not believe in counter-irritants in these, or in fact in any, cases.

In very high degrees of M. the *full* correction, even for distance, should not be ordered.

A single eye-glass should never be worn for myopia, except possibly for very occasional use in low degrees. It encourages monocular vision, and is exceedingly apt to lead to divergent strabismus in the uncorrected eye. Worn constantly, as it is by many Army officers, it is certainly harmful.

REGULAR ASTIGMATISM.

Astigmatism is that condition in which the refraction of the meridians of the eye is unequal. One meridian may be emmetropic, and the other myopic or hypermetropic (*simple* M. as. or H. as.) ; or both may be myopic or hypermetropic, but of different degrees (*compound* M. as. or H. as.) ; or one may be myopic and the other hypermetropic (*mixed* as.).

Objects looked at by an astigmatic are therefore not only indistinct, but distorted ; a circle becomes an ellipse, a point a line, etc.

An astigmatic whose horizontal meridian is emmetropic and the vertical ametropic will see a vertical line clearly, but a horizontal one as though its edges were "frayed out." This often puzzles beginners, who imagine the reverse should be the case, but the reason

is quite simple. If we assume each line to be composed of an infinite number of points, we can then realise that the points forming the *horizontal* line will be prolonged into short vertical lines by the *vertical* (ametropic) meridian; but the similar vertical lines made from the points composing the *vertical* line will be super-imposed, so that the line will be quite clean edged (though it will look a little longer than natural owing to the top and bottom points being converted into strokes). A little experiment with the aid of the accompanying diagram (Fig. 14) will make this ex-

FIG. 14.—RETINAL IMAGES IN REGULAR ASTIGMATISM. (FUCHS.)
A, two lines placed perpendicular to each other; B, their image upon the retina of an astigmatic person.

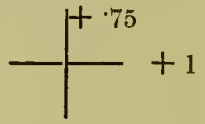
planation clear, and once grasped it will never be forgotten.

Look carefully with the naked eye at A; then place a − 1 cylinder, axis horizontal, before the eye (thus making the vertical meridian myopic, and leaving the horizontal emmetropic). You will now find A altered to B. Turn the cylinder round till the axis is vertical; you will then find the *vertical* line is the one blurred, while the horizontal is clearly marked.

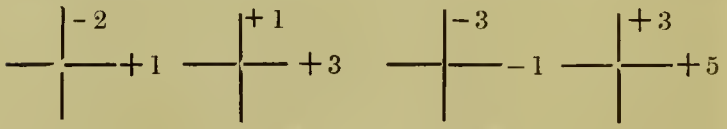
Astigmatism most often affects both eyes; when it does the direction of the principal meridians is probably the same in both. In high degrees of As. the visual acuity is usually reduced.

In most eyes there is a slight difference in the re-

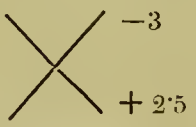
fraction of the meridians, the refracting power of the horizontal being generally less than that of the vertical; so long, however, as this difference does not exceed ·5 or even ·75, the eye may be considered normal. For example, the retinoscopy of an emmetropic eye (concave mirror—unmodified) may be :—



Compare this with the following retinoscopies (concave mirror—uncorrected) illustrating various forms of regular As. :—



Simple M. as. Simple H. as. Compound M. as. Compound H. as.



Mixed As. (oblique merids.).

As will be seen by these examples, the meridians, by crossing at right angles, form a cross which is generally vertical, but may be oblique.

Regular astigmatism is *caused* by a congenitally deformed cornea; astigmatism due to cicatrices in the cornea or sclerotic or sub-luxation of the lens being almost invariably irregular.

Estimation and Correction.—An example or two will serve to illustrate this better than pages of elucidation. In addition to reading these examples carefully with a trial case beside him, the surgeon would do well to visit the workshop of a friendly optician so as to watch the process of manufacture of cylinders and combinations of cylinders with spheres. The retinoscopies in the examples are supposed to be carried out with the concave mirror and corrected.

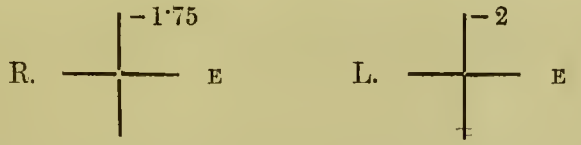
Case 1. *Simple M. as.*—Both near and distant vision

indistinct; patient notices that some letters are more distinct than others. On examination we find :—

$$V. = \text{R. } \frac{6}{18} \text{ partly: not imp. by sph.}$$

$$\text{L. } \frac{6}{24} \text{ partly: do.}$$

We do not waste time over fallacious *subjective* tests, such as clock-faces, radiating lines, etc., or putting up cylinders, but proceed at once to retinoscopy, and find :—



The theoretical correction should then be R. −1·75 cyl. ax. hor. : L. −2 cyl. ax. hor. When these are tried at the test case with the types, however, they are not quite satisfactory; patient prefers and reads $\frac{6}{6}$ with (each eye) :—

R. −1·5 cyl. ax. hor. L. −1·75 cyl. ax. hor.

We therefore order these as "spectacles for constant use." (The *pince-nez* form is not satisfactory for cylindrical lenses.)

Case 2. *Compound H. as.*—Patient, age fifteen, complains much in the same way as Case 1. His near vision is particularly bad

$$V. = \frac{6}{24} + 1 = \frac{6}{18} \text{ partly. R. and L.}$$

Retinoscopy (atropine) + 2 / + 4 R. and L.

Ordered the following glasses, which have been found satisfactory at the types :—

$$\frac{+ 2 \text{ sph.}}{+ 2 \text{ cyl. ax. vert.}} \quad \text{R. and L. Constant use.}$$

This prescription of combined spheres and cylinders needs a little explanation. The H. found (all of which it is found advisable to correct in this case) is + 2 in the vertical, + 4 in the horizontal, meridian. The + 2 *sphere* corrects, therefore, the vertical H. and + 2 of the horizontal; there remains +2 of the horizontal to be corrected, and hence we order a + 2 *cylinder* with its axis vertical, *i.e.* coinciding with the corrected meridian, and its curved part coinciding with and correcting the horizontal meridian.

Case 3. *Mixed As.*—Patient, a girl of twelve, cannot see the blackboard at school, and has great difficulty in sewing and reading; has been noticed putting her head on one side in looking at objects.

$$V. = \frac{6}{60}$$ R. and L. not imp. by sph.

Ordered :—

R. + 2 sph. / −4 cyl. ax. hor.

L. + 2 sph. / −3·5 cyl. ax. hor.

} V. with these $\frac{6}{9}$ each eye.

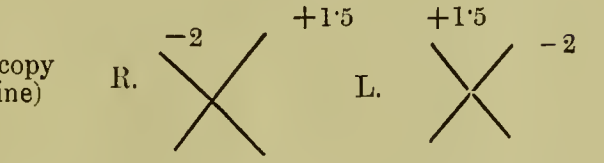
Here it will be observed that the + 2 sphere which corrects the horizontal H. increases the M. of the vertical meridian by 2 D, which must be taken into account when prescribing the cylinder. Various other combinations were tried at the final test with the types and test case, but no combinations enabled the patient to read $\frac{6}{6}$; this is an example of the reduction of visual acuity often present in As.

Case 4. *Mixed As.*—Another schoolgirl, complaining of very bad sight indeed for near and distant objects. In doing the retinoscopy the shadows in this case are

found to be *oblique*, and we roughly indicate the obliquity in the diagrams.

V. $= \frac{6}{60}$ barely, R. and L., not imp. by sph.

Retinoscopy (atropine)



Ordered :— $\frac{+ 1{\cdot}5 \text{ sph.}}{-3{\cdot}5 \text{ cyl. axis } 160°}$ R. and L.

(Vision with these is only $\frac{6}{12}$ but will probably improve.)

In order to explain the nomenclature of the cylinder in the above prescription the "trial-frame" worn by the patient while being tested must be carefully examined. The grooved half-circles into which we drop the trial-lenses will be found divided off into degrees by numbers from 0 at the inner angles to 180° at the outer. We already knew from the retinoscopy that the meridians were oblique, but we can only find out the exact degree of obliquity when we proceed to put up cylinders in the trial-frame later on in fitting the glasses with the aid of the types. We then find that the patient sees best with the axis of the cylinder not quite horizontal but inclined a little down and out; and on reading off the number on the trial-frame to which the axis corresponds, we find it to be 160°. Hence we order as above.

In some trial frames the degrees are numbered from 0 on *each side* down to 90° in the middle; using these frames, the above cylinder would be noted as "axis 20° down and out." Either way is at once understood by opticians.

Astigmatism can also be quickly estimated by an expert ophthalmologist by means of the *direct method*. A particular vessel on the disc is picked out, and the lens of the ophthalmoscope with which it is seen best is noted as the refraction of the *opposite* meridian; another

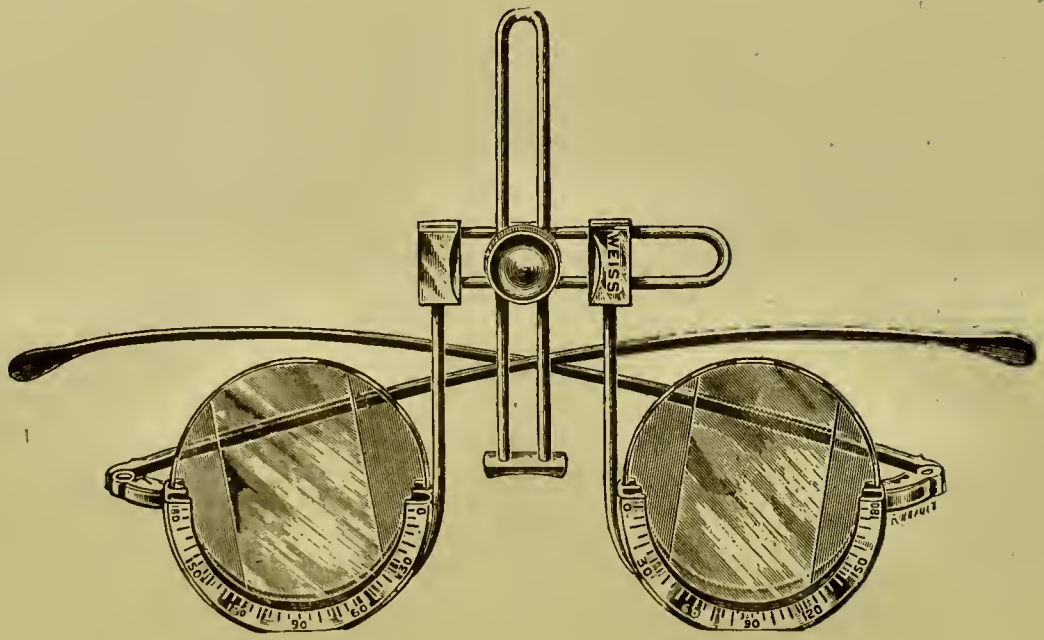


Fig. 15.—Trial Frames—with cyls. ax. 80° R., 70° L.

vessel more or less perpendicular to the former is similarly chosen, and the lens with which it is seen is taken to be the refraction of the other meridian. In fact, we regard the vessels as lines, and the explanation is the same as that of the clear vertical line and the horizontal blurred line given in the beginning. Much practice and no small amount of skill are needed in the direct estimation of astigmatism.

Javal's ophthalmometer, for the estimation of corneal astigmatism, is to be found in many eye hospitals, and a few consulting rooms. Mr. Swanzy uses one constantly at the National Eye and Ear Infirmary in Dublin. It is undoubtedly useful in quickly estimating astigmatism ; but it is cumbrous and expensive. In retinoscopy and direct ophthalmoscopy we have already two reliable methods (the latter very quick) of estimating, and I do not quite see the necessity of adding another.

Astigmatics requiring correcting cylinders (that is all except very slight cases) should not be passed into the Army. Even if the wearing of correcting glasses by the rank and file were generally permitted, I do not think an astigmatic recruit should be taken. Our Army has to serve in all parts of the world, and it would often be impossible to replace within a reasonable space of time broken cylindrical lenses.

Astigmatism and all other errors of refraction should be carefully looked for and corrected in soldiers' children ; for this purpose medical officers should visit the schools in barracks periodically during school hours, so as to observe the scholars at their work.

IRREGULAR ASTIGMATISM.

When the curvature of the *same* meridian varies, the condition is known as *irregular* astigmatism. Irregular astigmatism is present in everyone to a certain extent, inasmuch as the sectors of the lens differ slightly in their refracting power ; this is the reason why a star appears as a point with rays instead of as a simple point. In commencing opacity of the lens this natural astigmatism becomes exaggerated, and we get irregular (lental) astigmatism ; another example of lental irregular astig-

matism is that produced by sub-luxated lens. Corneal irregular astigmatism is due to changes in the shape of the cornea; it is most marked in "conical cornea," but may also be produced by nebulæ, ulcers, and operations.

It is often impossible, and always difficult, to correct irregular astigmatism by glasses. A prolonged and conscientious attempt should first be made to improve vision by various glasses selected empirically from the trial case; if this fail, "stenopæic" glasses may be found of service. Stenopæic glasses are simply blackened metallic discs, each with a circular or linear slit; various discs are tried, until the one best adapted to improve vision is found.

ANISOMETROPIA.

Anisometropia means a difference in the refraction of the two eyes. Slight degrees of this (see Astigmatism, Case 3) are very common. An extreme degree is seen when the lens of one eye has been removed, the other being normal. Many anisometropes are quite unaware of the difference between their eyes, just as "amblyopia" in one eye is often only found out accidentally; in cases where one eye is emmetropic or hypermetropic and the other myopic the patient unconsciously uses the former for distant, the latter for near, vision, and perhaps never suspects that he has not binocular vision.

Binocular vision may exist in anisometropia when the difference in refraction is slight; where, however, the difference is considerable one eye does all the work, and the other (non-working) eye is very apt to converge or diverge. One or other form of strabismus is found in about 25 per cent. of cases of anisometropia coming for treatment.

The correction of low degrees (say under 2 D) of anisometropia does not, as a rule, give much trouble; lenses are ordered correcting the refraction of each eye, and, after perhaps a little discomfort for a day or two, are "accepted," and good binocular vision obtained. In higher degrees the difficulty of obtaining binocular vision with each eye corrected is greater, and in such cases many surgeons either give the same glass for each

eye, or correct one eye only, putting plane glass before the other. This certainly saves both the surgeon and patient a good deal of worry, but is not fair to a patient to whom we may be able to restore binocular vision at the cost of a little trouble on both sides. Cases have been recorded in which differences of 10 D and more have been corrected, and binocular vision obtained. Quite recently I ordered glasses R − 8 L. − 4 to a brother officer; he had annoying diplopia for three days, but persevered, and has now clear binocular vision. Dr. Duane, of New York, puts the matter very clearly [1]:—

"My experience leads me to believe that in all cases in which the vision of the more ametropic eye can be brought up to anything like the normal *the attempt, at least, should be made to correct the anisometropia, and that this attempt should be persevered in* until either its success or its failure has been fully demonstrated." (The italics are his.)

The cause of failure in applying correcting glasses in anisometropia are supposed to be (Duane, *op. cit.*):—

(*a*) The image formed on one retina being larger than that on the other, satisfactory fusion cannot result. But if so, this is rather in favour of correcting, as in uncorrected cases the images are always of a very different size.

(*b*) Difficulty in wearing correcting glasses, especially strong cylinders. But this is because they are strong, not because they differ from each other. The difficulty will be remedied by use.

(*c*) Prismatic effect. In looking through anywhere but the centre of the glasses, patient looks through prisms, one stronger than the other. But if the glasses be put very close to the eyes (and if the patient be taught to move his head in the direction he wishes to look—M.T.Y.) this is minimised.

(*d*) Where there is diplopia from muscular deviation, but one image is practically neglected owing to its vagueness, the correcting glasses make both images clear and render the diplopia evident and annoying. This is serious; but even this will probably disappear with persistent use.

[1] "Knapp's" Archives of Ophthalmology, Nov., 1901; p. 637.

PRESBYOPIA.

We have seen that in an emmetrope the near point recedes beyond 22 c.m. at forty-five, owing to gradual loss of elasticity of the lens. About forty-four an emmetrope begins to notice he requires a strong light to read comfortably by (as it contracts the pupils), later he remarks that he has to hold small print daily farther and farther away; and at last he consults an oculist, or more frequently an optician, saying he wants what he calls "clearing" glasses for reading. I have often thought that practising ophthalmic surgeons become presbyopic earlier in life than others owing to the habit of relaxing the accommodation in direct ophthalmoscopy.

An emmetrope will require for near work +1 glasses at forty-five, + 2 at fifty, + 3 at fifty-five, +4 at sixty, + 4·5 or 5 at later ages. If he is wise he will even begin wearing glasses before he reaches forty-five—say + ·5 at forty-two, and + ·75 at forty-three.

These rules are subject to variation according to the work the patient is engaged on; a carpenter, for instance, unless he asks for *reading* glasses, should be allowed to choose such convex lenses as he finds best suited for his work. Reading glasses should enable the wearer to read Snellen ·5.

Myopes and Hypermetropes must be ordered presbyopic glasses on the same principle, making the necessary additions or subtractions; thus a myope of 2 D will not need glasses for near work till after fifty, and then begins with + 1; whereas a hypermetrope of 2 D will require them before forty.

When a presbyope begins to complain of his glasses becoming "too strong," and asks for weaker ones, incipient cataract should be suspected, as that disease often increases the refraction of the lens in its early stages; if, on the other hand, stronger and stronger glasses be asked for at short intervals, the possibility of glaucoma should be considered. (*Vide infra*, Cataract and Glaucoma.)

In *cycloplegia*, or paralysis of accommodation, due, apart from atropine, to many diseases such as diphtheria, influenza, etc., such convex glasses may be ordered as are found necessary to enable the patient to read until the improved tone of the ciliary muscle makes them unnecessary.

Spasm of accommodation has been already alluded to

CHAPTER V.

STRABISMUS, CONCOMITANT AND PARALYTIC. MALINGERING.

STRABISMUS, or squint, means a deviation in the direction of the eyes so that the visual axis of one is not directed on the fixed object. In *concomitant* squint the affected eye retains exactly the same relation to the other, no matter in what direction the eyes may be moved ; in *paralytic* squint—in which one or more of the ocular muscles are paralysed—the deviation is only evident within the sphere of action of the paralysed muscle. We can easily ascertain which form of squint we have to deal with by getting the patient to follow the movement of a finger up and down, in and out : in concomitant squint both eyes will follow the finger, strictly preserving their relative positions ; in paralytic, however, one eye will stop when the finger is moved in a particular direction, and then only will the squint become evident.

Concomitant Strabismus.—Having decided that a particular case is one of concomitant squint, we have next to find the deviating eye. Hold your forefinger close to the patient's face; ask him to fix it, and then cover rapidly one or the other eye with your hand or card. The deviating eye will then be recognised by the way in which it swings towards the object, the sound or fixing eye, on the contrary, remaining immobile.

Primary deviation is the deviation of the squinting eye. *Secondary* deviation is the deviation observed in the sound eye when it is covered by a card, and an object fixed by the squinting eye. In concomitant squint these deviations are equal, while in paralytic the secondary exceeds the primary.

When the same eye is always the squinting one, the

strabismus is known as *constant*, or monocular; when the two eyes squint alternately, *alternating;* when the squint comes only at intervals, *periodic.*

Theoretically a squinting person should see double, and in paralytic strabismus this double vision or diplopia is the most troublesome symptom. As a matter of fact, however, diplopia is rarely complained of in concomitant strabismus, because the individual affected gradually learns to exclude or suppress the image seen by the squinting eye, after the same fashion as microscopists and ophthalmoscopists learn to exclude the image on the eye not in use. A squinting person is often greatly helped in this exclusion by the fact that the visual acuity of the squinting eye is feeble; in fact, it is probably safe to say that the majority of squinting eyes are amblyopic.[1] Many believe that the retina of a squinting eye gradually loses its sensitiveness by disuse, and reasoning from analogy this seems at least probable (amblyopia ex anopsia).

It is of great importance from the point of prognosis and treatment to measure the extent of a squint pretty accurately; and the most reliable way of doing this is by measuring its *angle*, *i.e.* the angle the visual axis of the squinting eye makes with the normal axis, at a perimeter. This *angular method* is easily carried out:—

(*a*) Near Vision.—Fix the patient with the squinting eye at the centre of the perimeter, placing the arm of the instrument horizontally. Make him look with both eyes at the white spot in the centre of the instrument at the inner end of the arm. Now carry a lighted candle from within out along the arm until the image of the candle reaches the centre of the cornea of the squinting eye; then read off the number on the perimeter arm corresponding to the point the candle is held at. This, though it gives the direction of the

[1] "Amblyopia" has been humorously defined as a condition of the eye in which "neither the patient nor the surgeon can see anything." It is a convenient term meaning defect of vision without discoverable lesion. "Amaurosis" is used in much the same loose but convenient fashion to signify loss of vision.

optic instead of the visual axis, gives the angle of the squint sufficiently for all practical purposes.

(*b*) Distant Vision.—The patient should look at an object 6 metres away in a line behind the centre of the perimeter ; otherwise the procedure is the same.

In "Maddox's rod test" we have an excellent means of detecting slight squint in which the image seen by the affected eye is suppressed, and of detecting "heterophoria," or defective muscular equilibrium of the eyes. The disc containing the glass rod is to be found in many trial cases. Both the explanation and practice of the test are easy. The patient looks at the flame of a candle or gas jet, the disc containing the rod being placed, with the rod vertical or horizontal, in front of one eye. The effect of the glass rod is to apparently lengthen the flame, thus making the image seen by one eye so different from that seen by the other that there will be no impulse to unite them. If the lengthened streak of light then passes through the flame proper as thus seen, there is no diplopia, squint, or heterophoria. On the other hand, when latent diplopia is present the streak is seen above or below the flame if the rod be vertical (the direction of the streak is at right angles to the rod), on either side when it is horizontal.

Concomitant squint is caused by reduced acuteness of vision with disturbance of muscular equilibrium (heterophoria). The reduction of visual acuity causing squint is generally due to errors of refraction affecting one eye alone, or one more than the other. It may, however, be due to intraocular disease ; for instance, a blind eye often squints.

Concomitant squint is very common in our Army, owing to the large proportion of men with refractive errors who succeed in passing the very low vision test, and cannot or will not wear correcting glasses.

Convergent Concomitant Strabismus.—About 75 per cent. of all convergent squints are due to hypermetropia (including hypermetropic astigmatism and hypermetropic anisometropia). This is due to the intimate connection between accommodation and convergence. The hypermetrope has to employ a certain amount of accommodation even for distant objects ; as the object is brought closer

a still greater amount—with a corresponding amount of convergence—is used ; finally a point is reached when in exercising sufficient accommodation to render the object distinct too much convergence takes place, and one eye turns in. We can easily exhibit this cause and effect to a class by means of Donders' experiment :—

"We cause a man who has perfect muscular equilibrium in his eyes to fix his gaze on an object near by. If now we cover one of the eyes, it remains fixed in the correct position behind the screen. But if we now place a concave glass before the uncovered eye, so that the person under examination is compelled to accommodate quite strongly in order to see the object distinctly, the covered eye at once turns inwards—that is, an artificial convergent strabismus is produced, owing to the increased effort of accommodation."

It does not by any means follow that all hypermetropes squint. A man may find, for instance, that turning in one eye in extreme effort of accommodation produces diplopia ; instinctively then, in future he will avoid this extreme accommodation, and content himself with indistinct vision. In another case diplopia does not result from a similar effort, either because the hypermetrope "excludes" the image seen by the squinting eye or because the image is so indistinct, owing to defect or disease of that eye, that it is unnoticed — hence he squints. Again, squint is unusual in very high degrees of H. because the patient finds through experience that no effort of accommodation produces clear vision, and hence gives up the attempt.

The squint so often seen in very young infants is usually due to heterophoria, the child not having yet learned to co-ordinate the muscles of the two eyes, and soon passes off. True convergent squint due to H. does not come on till later, when the child first wishes to investigate near objects. Mr. Hartridge believes this form first makes its appearance about the fifth or sixth year, but my own experience leads me to believe that in the majority of cases it comes on much earlier, often as early as the beginning of the third year.

As a mother always has what she considers conclusive evidence as to the cause of a "birth-mark" in her child

so also is she usually firmly convinced of the truth of some equally remarkable theory to account for squint, such as that the child imitated another, that the nurse made him look too much in one direction, etc. It need hardly be said that these maternal theories as to the ætiology of squint are quite unfounded.

Divergent Concomitant Strabismus. — This is as common in myopes as convergent is in hypermetropes. The myope makes but little use of his accommodation, except for some very near object, for distant objects are naturally focussed in front of his retina, and near objects, unless very near indeed, require very little exercise of accommodation to produce clear images. As he uses little accommodation, so he will use little convergence ; apart from which, the increased length of his eye renders convergence difficult. Hence when convergence becomes a necessity, as for very close, minute work, his internal recti soon tire ; and at last, if the work be continued, one internal rectus gives up the effort, ceases contracting, and the corresponding eye deviates out. This divergence, at first only periodic, is apt to become permanent if the work be persisted in. Other causes of divergent concomitant squint are high anisometropia (including artificial anisometropia produced by wearing a single eyeglass) and too free operative interference in convergent strabismus.

It is evident from the above that divergent concomitant squint is due rather to weakness of the internal than to excessive action of the external rectus.

Treatment of Concomitant Strabismus.— *Convergent.*—In view of the fact that H. is the cause of the great majority of convergent squints, one would naturally imagine that correcting the error of refraction would cure the squint. Unfortunately, this is only the case in an extremely small and limited class of squints.

In quite small children with *periodic* squint, I have seen a successful result from the following procedure :—

(*a*) Correction of the *total* H. or H. as. by suitable glasses. Here it may be remarked that it is very difficult to induce a child under four or five to wear glasses constantly ; and even when worn, the child will often, if not carefully watched, develop a trick of looking over or under or any way but through them. Hence the co-

operation of intelligent parents or guardians is indispensable.

(*b*) Paralysing the accommodation by atropine. The drops should be instilled three times daily for a week, twice daily for a fortnight, once daily for a month, and thereafter once or twice a week for nine or ten months.

(*c*) For the first three months of the year's treatment a bandage should be worn for an hour daily over one eye—the one which usually fixes—so as to train the squinting eye in fixing.

Glasses, of course, must be worn much longer than the year, but can generally be gradually reduced in strength as the little patient "grows out of" the H. ; possibly in time they may be altogether dispensed with. A stereoscope, fitted with high convex lenses instead of prisms, forms a most useful adjunct to this treatment, and can be made into a most amusing and interesting plaything for a child ; but the elaborate "orthoptic exercises" devised on this principle, and described in books, present almost insuperable difficulties in carrying out, and are out of the question in Army practice.

The above treatment is only successful in a small number of cases ; still it is always worth trying in periodic squint, or possibly a very slight degree of permanent squint, in the very young. As a rule the internal rectus is actually shortened in permanent squint, and when that is the case non-operative treatment is simply waste of time.

Hirschberg's division of squints into five classes in respect of treatment is essentially sound, though it is difficult to lay down absolutely rigid rules. I have slightly modified it (adding a sixth class, etc.), as follows :—

1. Less than 10°—No operation ; *vide supra*.
2. „ „ 15°—Internal tenotomy of squinting eye.
3. „ „ 20°— „ „ both eyes.[1]
4. „ „ 30°—Internal tenotomy, and moderate advancement of external rectus, of squinting eye.
5. „ „ 45°—Ditto, but strong advancement,

[1] *Vide infra*, however.

and later internal tenotomy of the fixing eye.

6. Less than 60°—Double internal tenotomy; very strong advancement in squinting eye.

I am convinced that the sooner operative treatment is undertaken in cases of permanent squint of 15° and over the better. My friend Mr. Swanzy, in his well-known and valuable Handbook, lends the support of his great authority to the proposition that operative interference should, if possible, be postponed "to seven or eight years old," as "early childhood offers a decided obstacle to the careful adjustment of the operation and to orthoptic treatment." Personally, I think that operations for squint can be performed at much earlier ages with every prospect of success. I have little experience of the orthoptic treatment, but I do know that it would be very difficult to carry it out in soldiers' children or in the ophthalmic out-patient department of a large hospital. Even with private patients I can see no advantage in postponing active treatment; and if an oculist refuses to operate on a child until it reaches seven or eight years of age, the chances are the parents will take the patient to another surgeon who will operate at once, and probably with aggravating success.

Most squint operations, except perhaps simple tenotomy, require a combination of care, patience, and skill on the part of the operator that can only be acquired by experience. The vision and refraction of both eyes should be carefully estimated before operation, and full, or nearly full, correcting glasses worn after. As a matter of fact, however, the main purpose of the operation is a cosmetic one, as we can seldom hope to restore binocular vision.

Taking the cases, then, in accordance with Hirschberg's classification, we *may* think it worth while to try the atropine and full correction treatment, supplemented perhaps by orthopic exercises, in Class 1.

Coming to Class 2—cases of from 10° to 15° angle. Here *tenotomy*, or division of the tendon, of the internal rectus of the squinting eye, should, if thoroughly per-

formed, be sufficient for cure. For this little operation a general anæsthetic will probably be found necessary in a child under twelve ; after that age thorough cocainisation (by instilling cocaine every five minutes for twenty minutes before) is usually sufficient. The aim of the surgeon should be to leave a certain *slight* amount of convergence behind, as if the squint be absolutely corrected at the time of operation divergence may ensue later. As in all other eye operations, the eye should be thoroughly irrigated with a weak perchloride solution just before.

The eye speculum is now inserted. The surgeon should stand in front of the patient, whether the right or the left eye is to be operated on. A fold of conjunctiva and Tenon's capsule must then be seized by fixation forceps held in the left hand, just over the lower border of the insertion of the internal rectus tendon—about 4 m.m. from the cornea and just above a horizontal line drawn through the lowest point of the cornea. It is always best to press the closed forceps first firmly against the eyeball, then open it, and, *still* pressing, grasp a good thick fold between the blades. Professor Knapp rightly lays stress on the usefulness of this little manœuvre ; without it beginners are very apt to take up an insufficient fold of conjunctiva, with the result that they have to dig and poke about in the most awkward fashion when subsequently trying to hook up the tendon. A hole is now snipped in the fold by scissors held in the right hand ; the scissors are then passed in closed, opened, and made to snip a clear space all round the hole. Letting the scissors drop on to the little and ring fingers of the right hand, a strabismus hook is taken up by the thumb and forefinger of the same hand. The hook is inserted in the opening with its point and concavity *downwards*, embracing the eyeball so to speak ; it should then be swept upwards *towards* and always touching the eye, and so under the tendon. The forceps are now laid aside, and the hook holding the tendon transferred to the left hand ; then the right hand, taking the scissors again on the thumb and second finger, passes one blade under and the other over the tendon beneath the conjunctiva, and

snips through the tendon till the hook can be moved loosely under the conjunctiva right up to the cornea. The hook is now taken out, re-inserted, and made to catch up any outlying strands or expansions of the tendon which have previously escaped. No stitch is needed for the conjunctival wound unless the effect of the tenotomy seems excessive, when a "tuck" taken in the conjunctiva will lessen it.

The above is the "sub-conjunctival" operation, first performed by George Critchett, and generally known as the "Moorfields operation." Some operators, Mr. Silcock, of Moorfields, amongst the number, take exception to it, and prefer to lay bare the tendon, as they believe it is difficult to cut the tendon through completely under the conjunctiva; but I am bound to say that I could never convince myself that this objection is well founded. Needless to say, every care should be taken to insure that the tendon, *with its expansions*, is cut through by reinserting the hook and seeing that it can be carried freely right up to the cornea.

Immediately after the operation, if no anæsthetic has been used, otherwise after the return of consciousness, the eyes should be examined and the effect of the operation ascertained. If the effect be insufficient, an advancement of the external rectus of the same eye or tenotomy of the internal rectus of the other may have to be done later; if excessive, a tuck in the conjunctiva over the tendon will probably remedy it. Professor Knapp lays down the following useful rules :—

"After a tenotomy of the internal rectus the result of the immediate examination should be as follows :

1. In addition, the medial margin of the cornea should readily reach the caruncle.
2. The near point of binocular fixation should not be less than 5 c.m. ($2\frac{1}{2}$ inches).
3. The eyeball should not protrude.
4. There should be some convergence left."

If the effect produced be markedly excessive, it may be necessary not only to tuck the conjunctiva but to pass the needle very carefully through the episcleral tissue.

A pad and bandage should be worn over the operated eye for three or four days; then the full correcting glasses over both.

Tenotomy is a trivial operation, quite free from danger. There may be a little bleeding when the scissors snips through the sub-conjunctival tissue or through the tendon, but it is never serious. Subsequent inflammatory reaction can only be due to sepsis, which nowadays should be out of the question.

The next class in the division we have adopted is that of squints over 15° and under 20°. Here many operators perform an internal tenotomy of *both* eyes; a procedure which has, at all events, the merits of ease and simplicity. I am sure, however, that the principle of interfering, without absolute necessity, with the sound or fixing eye is a bad one, so that on the whole it is better to merge this class into the next.

In Class 4, squints between 30° and 40°, tenotomy being manifestly insufficient, we decide on the combined operation of tenotomy of the internal and *advancement* of the external rectus of the squinting eye. This operation takes a little time, and requires much care and patience. The surgeon must make up his mind beforehand as to the amount of advancement necessary, and yet may find he has to modify his first impression as the operation proceeds; and, as it is impossible to lay down precise rules to meet the varying circumstances of individual cases, I know of no operation in the whole range of ophthalmic surgery which requires more judgment and precision on the part of the operator, simple though its technique may be.

I believe it is best to perform the tenotomy separately, ten days or a fortnight before advancement.

To advance the external rectus:—

The patient is anæsthetised or cocainised, and the eye irrigated as for simple tenotomy; when cocaine alone is used it will be necessary to instil a drop or two pretty often in the course of the operation. Grasp the conjunctiva over the external rectus tendon, snip it through with scissors perpendicular to the axis of the tendon across the whole breadth of the muscle, and separate the conjunctiva on the corneal side

of the wound right up to the cornea; then clear the rectus tendon from the sclerotic by means of a strabismus hook passed under it. Laying aside the hook, the tendon is now grasped in a strabismus forceps, and cut across with scissors close to its insertion. Take up one of the double-needled sutures, pass one needle through the tendon near its upper corner, pull the thread half-way through, and lay both needles back on the side of the head. Another suture is passed in a similar fashion through the lower corner of the tendon, and laid on the cheek; and yet another through the middle of its edge, and laid on the ear.

Now take the unused needle of the upper suture in the holder, and pass it through the conjunctiva *and episcleral tissue* (this needs care) as close to the cornea as you think needful, and in the proper position as regards the upper margin of the cornea; do the same with the lower suture and the lower corneal margin, and then with the middle one and the middle of the corneal margin. Finally tie each suture with its own ends, first with a bow knot only, drawing each as tightly as necessary later when the final adjustment is made.

Mr. Stanford Morton has very kindly, at my request, furnished me with the following description of the operation of advancement by *folding*, or "tucking" (the expression is my own) the muscle on itself; an operation which I have repeatedly seen him perform with the happiest results at Moorfields:—

"Though differing somewhat in one or two minor details, the operation of advancing a muscle by folding it upon itself without division of its tendon is essentially that devised and largely practised by De Wecker, under the name of 'avancement capsulaire' (*vide* 'Annales d'Oculistique,' Tome 90, p. 188, 1883; also 'Arch. d'Ophtal.,' Tome 13, p. 1, 1893). This operation is perhaps specially adapted to those cases in which, with good vision in each eye, vertical or oblique diplopia might be produced if the tendon were divided and then became faultily attached. It may be performed separately, or combined with tenotomy of the opposing muscle when it is desired to increase the effect of this latter operation.

"The description here given applies to the operation as practised upon the external rectus (right eye).

"A vertical incision, three-quarters of an inch in length, is made through the conjunctiva an eighth of an inch from the outer corneal margin. The external

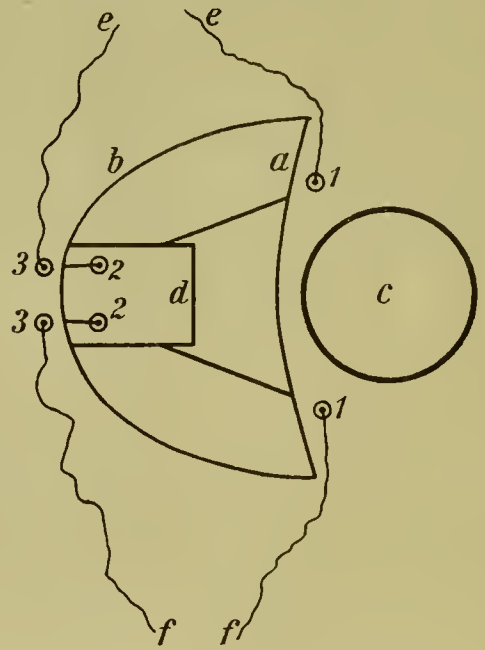


FIG. 16.—AFTER DE WECKER (MODIFIED), SHOWING THE STEPS OF THE OPERATION.

a, Inner edge of divided conjunctiva; *b*, outer edge of dissected back flap of conjunctiva and capsule; *c*, cornea; *d*, muscle; 1, entrance of suture into conjunctiva, capsule and sclerotic; 2, emergence of suture through muscle from beneath; 3, emergence of suture through capsule and conjunctiva; *e e* and *f f*, ends of the respective sutures.

portion (Fig. 16, *b*) of the divided conjunctiva, together with the capsule, is dissected back until the rectus (*d*) is freely exposed for fully half an inch. Blunt-ended scissors are then passed beneath the muscle to free its margins and under surface.

"The first suture is introduced at a point (1) a

little above the level of the upper margin of the cornea, and about in a line with its external border. A No. 3 small curved needle, armed with No. 1 plaited silk, is made to pick up just enough of the superficial fibres of the sclerotic to give a firm hold for the suture; if this object has been attained, then on gently lifting the needle the eyeball is slightly drawn forwards—otherwise the needle merely lifts the conjunctiva from the globe. On being satisfied by means of this test, the needle is brought out in the wound. The muscle being now gently raised on a strabismus hook, the needle is next carried across the wound and made to pass through the muscle from beneath at a point (2) generally about a quarter of an inch from its insertion, though this point naturally varies with the effect desired. Finally, the needle is brought out from beneath through the capsule and conjunctiva near the edge of the dissected-back flap (3).

"A second needle is passed in a similar manner on the other side of the horizontal meridian (1, 2, 3).

"The two sutures being now in position, if the opposing muscle is to be divided it should be done at this stage.

"On now tying the two ends (*e e* and *f f*) of each suture separately a fold of the muscle with its capsule is drawn as far as necessary towards the corneal margin. Each suture should be tied first with a double twist of the silk (first half of a 'surgeon's knot'), which can be relaxed or further tightened until the required adjustment is attained; then the 'surgeon's knot' is completed.

"Should it be necessary to perform the operation under an anæsthetic, it is a good plan to tie each suture in a bow knot with the ends sufficiently long to reach past the outer canthus to the temple where they are fixed with plaster. The adjustment can then be more satisfactorily effected after recovery from the anæsthetic.

"The effect produced can be somewhat increased by excising a strip, an eighth to a quarter of an inch wide, from the edge of the dissected-back flap of capsule and conjunctiva."

The operation of advancement, with or without

tenotomy of the opposing muscle, if carried out with skill and, above all, judgment, will cure the squint from a cosmetic point of view ; but in most cases it is very difficult, in many impossible, to restore binocular vision. Exercises with the stereoscope (orthoptic treatment) may be tried subsequent to the operation with this object in view, and are occasionally successful.

Classes 5 and 6 of convergent strabismus must be

FIG. 16*a*.—SHOWING SECTION THROUGH MUSCLE, ETC.

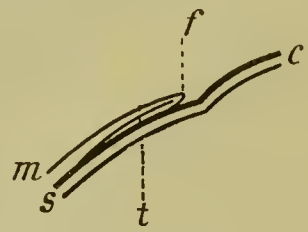


FIG. 16*b*.—SHOWING SECTION THROUGH MUSCLE FOLDED AT *f* IN ADVANCE OF ITS INSERTION (*t*).

dealt with by the same procedure, but in different degrees, viz. by tenotomy of the internal, and strong advancement of the external, rectus of the squinting eye, and later internal tenotomy of the other eye.

Divergent Strabismus.—Here the deviation out is caused more by weakening of the internal rectus than by undue action of the external rectus ; and operative interference—tenotomy of the external rectus—is not so likely to be attended with success ; or operation may, in a sense, be only too successful, leading to convergent strabismus.

On the whole, operation is not advisable except in carefully selected cases in which treatment by glasses

has failed. Correcting glasses will cure a certain number of cases; others may require correcting glasses decentralised (out in M., in in H.); others prisms, worn *base inwards* (inducing adduction to obtain single vision).

Diplopia in Concomitant Strabismus. – When diplopia is present (*vide supra*) in *convergent* concomitant strabismus, it is of the *homonymous* variety, *i.e.* the image of the object seen by the squinting eye is on the same side as that eye. Say a patient has left internal squint; if a lighted candle be held before him, a disc of red glass dropped in front of his left eye, two images of the candle will be seen, one red (the so-called *false* one, seen by the squinting eye) on the left of the colourless *true* image seen by the right or fixing eye. The explanation is simple: the right or fixing eye sees the candle with the macula; the left, squinting inwards, sees it with a part of the retina accustomed to seeing images on the left, and consequently *refers* the sensation to the left.

In *divergent* concomitant strabismus, on the contrary, the diplopia is *crossed*. In the previous experiment, if the left eye squint outwards the retina of that eye receives the candle-image on a part of the eye accustomed to sensations from the far right; hence, of the two candles seen, the red or false one is to the right of the colourless true one.

A convenient "memoria technica" may be embodied in the proposition: "When the visual axes cross (*e.g.* convergent strabismus) the images do not, and *vice versâ*."

Paralytic Strabismus.

In paralytic strabismus both eyes will follow a moving object, such as a pen or finger, up to a certain point; then one—the squinting eye—stops, while the unaffected eye still follows.

Paralytic strabismus is due to paralysis of one or a group of the extrinsic muscles of the eye; such paralyses are due to two sets of causes—(1) lesions within the orbit, or (2) lesions within the skull—and must, of course, be looked on only as symptoms. An exhaustive discussion of the causes of paralytic strabismus does not come within the scope of this work. It is proposed, therefore

only to give simple rules enabling the surgeon to ascertain what group of muscles, or what muscle of those most commonly affected, is paralysed in individual cases; the diagnosis and treatment of the cause of the paralysis belongs to the domain of general medicine and surgery.

All the muscles of the eye, including the intra-ocular, may be paralysed; the lid hangs down, the eye looks immovably forward, the pupil is widely dilated and the power of accommodation gone. This is known as *ophthalmoplegia totalis.* In *ophthalmoplegia externa* only the extrinsic muscles are affected; in *ophthalmoplegia interna* only the intrinsic (*e.g.* after atropine, in diphtheria, etc.).

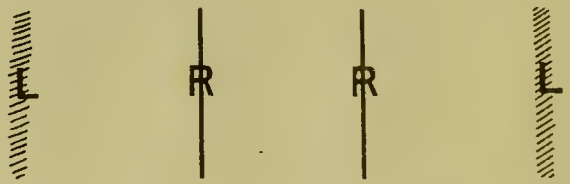


FIG. 17*a*.—LEFT EYE AFFECTED. FIG. 17*b*.—RIGHT EYE AFFECTED.

Except in the cases of the external rectus and superior oblique, which have each a separate innervation, paralysis of a single extrinsic muscle is infrequent (with the possible exception of the superior rectus—Duane).

Kling[1] analysed a large number of cases of paralytic squint with the following result:—

Paralysis of sixth nerve (abducens) in 52 per cent.
„ „ third „ (oculo-motor) in 45 per cent.
„ „ fourth „ (trochlear) in 2·5 per cent.

Paralysis of the external rectus (sixth nerve) produces convergent squint with homonymous diplopia; the lateral separation of the images increases as the paralysed eye is *abducted.* In the candle test, therefore, putting the red glass before the affected eye, the images will appear as in the figure (the shaded line represents the false image). (17*a* and 17*b*.)

In *oculo-motor paralysis* (third nerve) there is re-

[1] Inaugural Dissertation, Giessen, 1901.

markable and characteristic deformity. The lid drops, the eye is pulled *out* and *down* by the external rectus and superior oblique (so that the diplopia is crossed, the false image being slightly higher than and inclined to the true), the pupil is widely dilated, and accommodation gone ; there is generally some slight exophthalmous. A rare variety of this paralysis is known as "ophthalmoplegic migraine" ; the paralysis is intermittent, comes on as part of an attack of violent migraine affecting the same side of the head, and only lasts a few days.

Paralysis of the superior oblique (trochlear) is often not noticeable at a casual inspection, as the work of the muscle is more or less "taken over" by the internal and inferior rectus ; and diplopia is only found below the horizontal plane. On getting the patient to look down, however, he at once complains of diplopia ; the false image is lower than and tilted towards the true ; as the eye is depressed and adducted the vertical distance between the images increases, when abducted the obliquity of the false image increases.

The surgeon should adopt some such simple method of noting the variety of diplopia as is shown in the diagrams. The patient, with a red glass before one eye and none or a colourless one before the other, stands facing a large blackboard or dark wall ; a mark is made on the wall which he is directed to fix, while the surgeon moves the candle in various directions, horizontally and obliquely, round the mark until the positions in which the diplopia appears are detected and noted.

For further information on the interesting and intricate subject of paralyses of the orbital muscles the reader is referred to any of the larger systematic treatises on ophthalmology.

Nystagmus.

Nystagmus is not squint, but inasmuch as it is a disturbance of the mobility of the eye may be considered here. It is a very curious phenomenon, consisting of more or less continuous, very short, jerky oscillations of the eye, usually bilateral, ceasing during

sleep. According to the direction of the movement it is known as horizontal, vertical, oblique, or rotatory nystagmus. Strabismus frequently accompanies it, but apart from this the movement of each eye as a whole is unaffected.

It is most frequently a congenital defect, and is then invariably accompanied by some imperfection of the eye, such as albinism, involving defective visual acuity. Congenital nystagmus is attributed with great unanimity to the fact that the child has never learned to fix, owing to the ill-defined images of external objects seen by its defective eyes; but I fail to see how this would account, entirely at all events, for the peculiar rhythmic, involuntary character of the motion.

Nystagmus is also found as a symptom of certain lesions of the central nervous system, such as insular sclerosis, lesions of the optic thalami, hereditary tabes (Friedreich), etc., and is unfortunately by no means infrequent among coal-miners, especially "coal getters" or "pick men," who have to work in constrained positions of body and eyes (miner's nystagmus). The probable cause of miner's nystagmus is chronic weariness of the eye muscles leading to clonic spasms.

Miner's or "occupational" nystagmus usually disappears with rest from the occupation, though I have seen a case in a reservist in South Africa who had not worked in a mine for three years.

MALINGERING.

Malingering to avoid Army service is very common in countries where military service is compulsory; so much so that precise rules for its detection are inserted in the medical regulations of some foreign armies. It is generally assumed that malingering with this purpose is unknown in England, owing to our voluntary system of enlistment; this, however, is by no means the case. Certainly it is not very common; but most recruiting medical officers have seen cases in men who, for one reason or another, alter their minds as to entering the Army between the time they proffer themselves for enlistment and the time of medical inspection.

In our own Army, however, malingering is much

more usual in men who have been some time in the Service, and who hope, by simulating visual defects, to be invalided, and possibly, when the supposed blindness is attributed to injury incurred "in and by the Service," specially pensioned. Simulating blindness or defective vision with a view to obtaining legal damages is common enough, and often successful, in civil life.

Though I have seen many malingerers in the course of my service, I have never yet met a skilful one, or one who could not be detected in a few minutes by ordinary common-sense tests; nor have I ever seen a malingerer affect bilateral blindness.

Hysterical defective vision is seen occasionally in young girls; such cases were common enough in my time at the Moorfields clinic, and were occasionally bilateral.

Malingering may very reasonably be *suspected* if nothing abnormal can be detected in the eye or eyes on examination; but should by no means be *assumed* for this reason alone, for the patient may be amblyopic, and cases of congenital amblyopia do somehow manage to get into the Army through carelessness on the part of the examiner or trickery on the part of the recruiting sergeant. In regard to ophthalmoscopic examination it should also be remembered that malingerers have been known to produce mydriasis by means of atropine "drops" obtained from some chemist.

Simulated Bilateral Blindness.—The simulation of total blindness is extremely rare, as to carry out the deceit with any hope of success would require extraordinary dramatic instincts in addition to patience and cleverness. Schmidt Rimpler suggests testing in such cases by holding the patient's own hand in front of him, and asking him to look in its direction; the genuinely blind man will, of course, do this at once, but the malingerer will probably think he ought to look in a different direction. Even bilateral *defective vision* is seldom assumed. Careful inquiry into the history, thorough ophthalmoscopic examination, testing with lenses, and perhaps a suggestion of the urgency of operative interference, should generally reveal the true nature of such cases (*vide infra*—Prisms).

Simulated Unilateral Blindness.—If total blindness be alleged, it may for all practical purposes be considered to be simulated if the pupil of the supposed blind eye reacts to light, or if it reacts "consensually" with the pupil of the other; though there have been rare cases in which the pupillary reaction has been observed in a blind eye. The possible presence of synechiæ or artificial mydriasis should not be forgotten; but we are now assuming that the eye has been first thoroughly examined. As a rule, however, the malingerer affects only defective vision, or *aggravates* a pre-existing defect in the eye. The following are tests which I have found most convenient and serviceable in practice; in the Army, at all events, it is rarely necessary to go beyond the first. The suspected person should be carefully watched while he is being tested, as clever malingerers have been known to "give themselves away" by rapidly closing the alleged blind eye in an endeavour to ascertain what they should or should not see.

(1) The patient is placed before test types arranged in the ordinary way, but with the rows of letters alternately red and green. He is then asked to read the types with both eyes; he will probably read all or nearly all, as one eye is admittedly sound. A trial frame, with a red glass for one eye and a green for the other, is then put on his nose; the red letters only can now be seen through the red glass, the green through the green, for red and green are complementary colours. If now he can read the same as before, we have not only found that he is malingering, but have also estimated the visual acuity of each eye; if he only reads the red or green letters, not both, he is almost certainly blind in one eye unless he be a very expert malingerer. When the special types for this test are not available, letters written with red and green pencils on white paper may be improvised in their place. Should the malingerer have read all the letters, he may now be openly convicted and "shown up" by telling him to close the alleged blind eye.

(2) There are several pretty ways of detecting imposture by means of *prisms*. I need give two only.

We pretend to discover some defect in the admittedly

sound eye, having previously covered the supposed blind eye ; a candle is held in front, and a prism of about 15 is slowly moved up half-way across the pupil. The patient will now see double (and will not object to admit it, as he knows the other eye is covered), because while the rays above the prism pass straight in, those through it are refracted. Now the cover is taken off the other eye, and *at the same time* the prism is moved up so as to cover the whole pupil of the sound eye. He will almost certainly continue to say he sees double, whereas if the pretended blind eye were really blind he would see single. Even now we do not stop, but proceed to estimate the visual acuity of both eyes with types ; of the two images of the letters seen, the upper one is that seen by the blind (?) eye. The essential prettiness of this and the last test consists in the fact that the malingerer is exposed and the visual acuity of each eye ascertained at one and the same trial ; and nothing in a small way is more gratifying than the successful detection of imposture by their means.

Another prism test is based on the natural and practically irresistible desire for single vision. The person tested looks at a candle ; a prism, base inwards, is then held in front of the blind (?) eye ; if the eye sees, he will involuntarily *abduct* it in order to get the image on the same part of the retina as it is on in the other eye. For the same reason *adduction* will take place if the prism be held base outwards. The same test is applicable to simulated binocular blindness (Priestley Smith).

(3) A very quick and handy method is to pretend to try the effect of glasses on both eyes together. First, a couple of weak concave glasses, say — ·25's, are dropped into the trial frame ; he still reads the types clearly. Then a pair of — ·5's are put in ; he still reads easily. Having thus diverted his suspicions, we suddenly drop in, *at the same time*, a + 5 or + 6 lens before the sound eye and a weak minus or plane glass in front of the blind (?) eye. If now he reads the types, it is evident he is reading with the blind (?) eye, as he could not see them through the high *plus* lens, and he may be convicted at once by making him shut the eye behind the weak concave lens.

There are many other tests, some most elaborate, but the above are quite sufficient for our purposes. Those interested in the subject, which is both a fascinating and amusing one, will find the numerous procedures dilated on lovingly in Frolich's monograph.[1]

One note of warning in conclusion. Whatever may be the case in foreign armies, men are much more often *unjustly* than *justly* suspected of malingering in our own.

[1] Des procédés modernes pour reconnaitre la simulation de la cécité ou de la faiblease visuelle.—"Revue Medicale de la Sûisse Romande," 1891, p. 721.

Bar reading -

CHAPTER VI.

THE OPHTHALMOSCOPE IN GENERAL MEDICINE.

The fact that intraocular changes are present in many general diseases, and that they are frequently of the greatest diagnostic and prognostic importance, renders familiarity with the use of the ophthalmoscope a most necessary portion of the equipment of the practising physician and surgeon; and no class of general practitioner requires it more than the Army surgeon, whose *clientèle*, be it remembered, includes not only officers and men, but also their wives and families. As Dr. Gowers justly observes:—

"By the aid of the ophthalmoscope, we can observe magnified . . . the termination of an artery, of a vein, and of a nerve; a peculiar vascular structure (the chorioid); and a peculiar nervous structure, the retina. Nowhere else are nerve and vessels exposed to direct observation. Many changes affecting these tissues throughout the body may be *first and best* detected here, and in some other diseases these intraocular structures are affected in a special manner." The italics are mine. I only propose to indicate briefly here such characteristic intraocular, or rather fundus, changes as are potent factors in the diagnosis, prognosis, or treatment of general diseases. Foremost in frequency and importance amongst these are the changes observed in

DISEASES OF THE URINARY SYSTEM.

Uræmic Amaurosis.[1]—The word "uræmic" is a possibly inaccurate, and certainly incomplete, adjective employed to indicate a group of symptoms of renal

[1] Amaurosis is an unscientific but convenient term of the same class as amblyopia; but while the latter refers to *defective* vision only, the former means blindness—in either case without discoverable lesion.

disease of cerebral origin, including epileptiform seizures, headache, stupor, coma, vomiting, and amaurosis. The last-named symptom is the only one that will be discussed here.

Uræmic amaurosis is common enough, but as it forms but one of many symptoms of a very grave condition it is very apt to be overlooked, the physician contenting himself with noting the condition of the pupils, and then proceeding to deal with the graver general symptoms. It is generally connected with an epileptiform seizure; coming on as a rule after a convulsion, exceptionally before.

"The onset of the amaurosis is usually sudden, and the loss of sight bilateral and complete; even perception of light is abolished. . . . The duration of the blindness varies. Sight is usually regained in twelve to twenty-four hours, but occasionally not for two or three days. Recovery of vision is as a rule complete. Recurring attacks of amaurosis have been met with during the course of a case of renal disease, and in such it is said that permanent defect of vision may ensue." (Lawford: "System of Diseases of the Eye," by Norris and Oliver.)

In the majority of cases recorded by ophthalmologists, the reaction of the pupil to light persists; in a minority it is abolished. With the limited amount of scientific evidence at present available it does not seem yet possible to elevate this sign into a sure element of prognosis; but, speaking generally, it may be said that the patient is probably not in such immediate danger in the former case as in the latter. For if the pupil reflex be lost, the probabilities are that both the central and peripheral parts of the optic apparatus are poisoned by the noxious substances accumulated in the blood; if it be retained, the part above the corpora quadrigemina only is affected.

The only ophthalmoscopic changes recorded have been in cases of pre-existing albuminuric retinitis, in which increased swelling of the optic disc has been stated to have been found.

Visually the prognosis is good, complete recovery of vision within a few hours being the rule.

Uræmic amaurosis, like uræmia generally, has been noticed chiefly in the acuter forms of renal disease, more especially in the uræmia due to primiparous pregnancy and scarlet fever.

Renal Neuro-retinitis. — This nomenclature seems preferable to "albuminuric retinitis." The latter expression, though sanctified by usage, is not a very happy one—first, because renal disease with ocular changes may be present without albuminuria; and second, because the optic nerve is generally affected as well as the retina, and the old name gives no indication of this. The fact that the optic nerve is affected forms one of the distinctions between renal neuro-retinitis and "diabetic retinitis" (*vide infra*).

Mr. Marcus Gunn, under whose guidance I had the advantage of working for some years at Moorfields, has reduced the careful and painstaking inspection of the minuter changes in the fundus to a fine art; and we are fortunate in the fact that he has recorded some, at least, of his observations bearing on this important subject[1] so that the results of his luminous investigations need not be confined to the circle of his pupils. In the valuable paper alluded to he describes and discusses the intra-ocular evidences of that general arterio-sclerosis which is characteristic of the subjects of contracted granular kidney—the special form of renal disease with which neuro-retinal lesions are chiefly associated. Needless to say, it is of the greatest importance to the practical physician to be able to detect these vascular changes, which, being antecedent not only to the graver neuro-retinal changes but also to the usual evidences of renal disease, act as valuable "danger signals," and afford reliable indications for preventive treatment.

These changes antecedent to the neuro-retinitis include alterations in the arteries, mechanical pressure on the veins, and œdema of the retina.

1. Changes in the arteries. (*a*) The arteries are abnormally and irregularly *narrowed*. An artery of apparently normal size suddenly narrows, continues narrow for some distance, and then regains its original breadth; or there may be a local increase of breadth,

[1] "Transactions of the Ophthalmological Society," 1898.

the artery tapering off at each extremity to normal or sub-normal size — specially well-marked when it occurs in the macular region, where a bright red vessel of unusual size at once attracts notice. (*b*) They are also often very tortuous. (*c*) *The central light streak is very bright*, this being most noticeable (unlike the light streak in high H. or optic neuritis) in the *smaller* branches ; and the entire breadth of such arteries seems abnormally lighter in colour. (*d*) Loss of translucency. An underlying vein can no longer be seen through the artery as in the normal eye.

2. Mechanical pressure on the veins. The hard, diseased artery soon shows the influence of its pressure on an underlying vein ; at first but slightly, but later by markedly impeding the flow of the venous current.

3. A want of the full red fundus-reflex, amounting occasionally to a decided grey haze, most marked at the macula, is very commonly present.

Unfortunately, patients in this *pro-dromal* stage of renal neuro-retinitis do not often come under the notice of the ophthalmic surgeon ; and physicians are only too apt to forget the valuable information they may derive from an ophthalmoscopic examination in cases of suspected renal disease—more especially in cases where no albumen can be detected, and the suspicion rests mainly on the lowered specific gravity of the urine. Here again the expression "albuminuric retinitis" is a most unfortunate and misleading one.

Neuro-retinitis is most frequently found in chronic cases of renal disease of the "contracted granular kidney" type, though it is seen fairly often in the albuminuria of pregnancy, and very occasionally in acute kidney trouble. Cases of the latter variety, however, will generally be found, on closely examining the history, to be exacerbations of pre-existing disease.

"The period of the renal disease at which retinal changes develop cannot easily be determined. It is now fairly well established that the retinal lesions never appear prior to evidence of disease of the kidney, as was formerly supposed. It is true that retinal changes may be met with in patients with granular kidneys but without albuminuria at the time of the onset of the

retinitis. Such instances are, however, uncommon, and are cases in which the albumen is present intermittently, or does not appear till a very late stage of the kidney disease." (Lawford, *op. cit.*)

On the whole, retinal complications are rare during the first six months of the renal disease. Renal neuro-retinitis is almost invariably bilateral, and often very symmetrical. Although a somewhat late symptom of kidney disease, it often happens that it is the first noticed by the patient; so that here, as in locomotor ataxy, the general disease is often first detected by an oculist. With one conspicuous exception, it is a certain sign of a fatal, and probably early, termination of the renal disease. The exception is the retinitis complicating the albuminuria of pregnancy; this does not appear to influence the prognosis one way or the other.

The classical ophthalmoscopic picture of renal neuro-retinitis is briefly as follows :—

The disc is pink, hazy, with blurred margins. The retina in its neighbourhood is greyish and hazy, with here and there red patches of hæmorrhage and white patches of degeneration. Around the macula are white streaks of degeneration radiating in stellate fashion from the dark fovea. The vessels present an advanced degree of the changes described by Mr. Gunn. In a more advanced stage still the macular region may be seen as a vivid white star figure, the disc so swollen as to be found with difficulty, and the whole fundus speckled with hæmorrhages, large near the disc, smaller at the periphery.

Such is the typical picture; but it is a picture susceptible of almost infinite variation, though two elements in its composition are present in almost every case—the neuritis and the hæmorrhages. Hence the wise rule followed by oculists of invariably examining the urine in cases of retinal hæmorrhages, a rule which might with advantage be extended to cases of optic neuritis.

In rare cases changes in the chorioid, hæmorrhages into the vitreous, and detachment of retina occur.

The loss of vision is by no means in proportion to the extent of the lesions; this disproportion is most

observable in the form associated with pregnancy, where almost complete restoration of vision frequently takes place, though the changes in the fundus may have been very extensive. Peripheral vision is often good, even in bad cases, and the light-sense is but little affected.

Prognosis, except in the case of pregnancy, is invariably grave. Patients with renal retinitis coming to ophthalmic clinics soon cease attending, for the best (or worst) of reasons. At Moorfields we found that amongst the class usually represented by out-patients the presence of renal neuro-retinitis meant death within six months. The only prognosis statistics on a large scale that I know of are those collected by the Baroness Possaner from Professor Haab's Zurich clinic and private practice. Schobl, of Prague, quotes the following data from her work :—

"Men of the poorer classes coming to the clinic die within two years—that is their percentage of death is 100—while among women of the same class the percentage is only 68. Of the private patients possessed of means the death-rate is 59 per cent. for men, and 53 per cent. for women. The longest period of life after the retinitis had been diagnosed was six years for the clinical, and eleven years for the private ones."

The treatment is the treatment of the general disease; no special eye treatment has the slightest value.

Cataract and paralysis of ocular muscles have been recorded in cases of Bright's disease, but were probably only coincidences (Deutschmann, Knies).

Diabetic Retinitis.—Cataract, by far the most common ocular complication of diabetes, hardly comes within the scope of the present chapter; it is probably due, as shown by experiment, to the absorption of moisture from the lens by the sugar-containing aqueous humour. Von Graefe found that it developed in 25 per cent. of diabetics. Its removal is said to be occasionally followed by diabetic coma, but on the whole the results of operation are pretty satisfactory. Next to cataract, the most frequent ocular complication is *diabetic retinitis*. The fact that in many cases of diabetes albumen is present as well as sugar for a long time

induced observers to attribute the retinal lesions seen in diabetes to the renal disease. There is now, however, little doubt that there is a specific diabetic retinitis, though there is an astonishing discrepancy in the statistics of its frequency collected by well-known ophthalmologists. Hirschberg, for instance, states it is rarely absent in diabetics of some years' standing, De Wecker believes it to be a very rare complication, and Seegen found but two cases in 140 diabetics. During the five years I was clinical assistant at Moorfields I only saw three cases in which the diagnosis was pretty certain; none of the three had albuminuria—one only, however,[1] was under my observation for any length of time.

In its most typical form—called by Hirschberg "central punctate diabetic retinitis"—small palest yellow or ivory-white plaques are found in the macular region and just within the curves formed by the superior and inferior temporal vessels; *fine* hæmorrhages are scattered throughout the fundus, largest and best marked above, below, and outside the macula; there is no swelling of the disc, no clouding of the retina. Most of the ivory spots lie in front of the vessels. Small extravasations into the vitreous are common. In the case which I recorded (*vide* footnote) the ivory spots had coalesced in the macular region, producing a very peculiar appearance resembling an optic disc (pictured in the Transactions); this patient has been under my observation now for over three years.

The chief distinguishing marks of diabetic, as compared with renal, retinitis are (1) unaltered disc, (2) no swelling of retina, (3) no apparent change in blood-vessels, (4) *fine* hæmorrhages, (5) patches of degeneration *dull* white and not stellate.

It is difficult, in view of the discrepancies in the records, to decide on the exact bearing of the retinal changes on prognosis; all that can be said with our present imperfect knowledge is that prognosis is not nearly so unfavourable as in renal neuro-retinitis.

[1] "Transactions of the Ophthalmological Society," vol. xix., 1899.

DISEASES OF THE BLOOD AND HEART.

In **anæmia** and **chlorosis** pallor of the disc and translucency of the blood-vessels may be present, but the conditions of the ocular circulation are so different from those of the general circulation that too much stress must not be laid on the presence or absence of these signs in a given case. In **pernicious anæmia** hæmorrhages, sometimes of large size, are frequently found, and seem to be invariably present in fatal cases. **Scurvy** and **purpura** pretty constantly produce retinal hæmorrhages. The picture of the fundus in **leukæmia** is a startling one ; the whole fundus is of a golden or pinkish orange colour; the disc swollen and indistinct, veins dilated and tortuous ; while numerous fine hæmorrhages and dull white stripes and spots are scattered throughout the entire retina.

Obstruction of the central artery of the retina by thrombosis or embolism (which is the more common pathogeny is still a matter of dispute) may occur in heart disease, chiefly, according to Gowers, in *mitral stenosis* cases, sudden and complete (*vide infra*, however) blindness being the result. The ophthalmoscopic picture is unmistakable. There is extreme pallor of the fundus corresponding to the whole posterior pole of the eye ; the arteries are reduced to threads, and the veins also contracted but not nearly to the same extent ; the disc is pale and ill-defined. At the fovea is a cherry-red spot, standing out from the surrounding pallor like a little red lamp ; this is generally supposed to be due to the effect of contrast, but it seems more than probable that there is a hæmorrhage into either the retina or the chorioid at this spot.

When the macula is supplied by a "cilio-retinal" vessel the macular area may remain unaffected and some central vision be left.

When only a branch of the central artery is obstructed the effects are confined to the area supplied by the branch, and the ensuing visual disturbance is a large or small scotoma corresponding to the affected part of the retina.

If the patient be seen at once, energetic massage of

the (previously cocainised) eye may shift the clot; and even if seen later this procedure may be tried. The clot is sometimes—very rarely, however—moved spontaneously; I have a vivid recollection of such a case occurring some years ago in Mr. Gunn's clinic at Moorfields.

Thrombosis of the central vein of the retina may be found in old cases of *heart disease*, *arterio-sclerosis*, and in *pregnancy;* the onset of blindness is not so sudden as when the artery is affected. The majority of cases have been found in peasants who work in a stooping posture (Schobl). The disc is swollen and blurred, the veins enormously engorged, the arteries scarcely visible, and the whole fundus covered with hæmorrhages. Treatment is useless.

Gowers describes peculiar retinal hæmorrhages—round, with pale or white centres—as almost pathognomonic of ulcerative endocarditis.

DISEASES OF THE BRAIN AND SPINAL CORD.

Double optic neuritis is a prominent symptom, frequently one of the first, in *cerebral tumour or abscess*, and is of great importance in the diagnosis of the brain lesion, as it is present in at least 80 per cent. of all cases; in fact, it is so clearly recognised as a sign of cerebral tumour that authorities on brain disease advise physicians to hesitate before committing themselves to a positive diagnosis in its absence. The most characteristic appearance this variety of optic neuritis assumes is that of "choked disc": here the "swelling of the papilla is great and abrupt and its margin defined, while the central artery is much diminished in calibre, and the central vein engorged" (Swanzy). Swelling of the optic disc is diagnosed by the fact that the top can be seen with a higher + lens than the retina at the base; each diopter employed means about $\frac{1}{3}$ m.m. in height, so that when the retina is seen without a lens and the top of the disc with + 3 there is swelling to the extent of 1 m.m. In a more uncommon form the margins of the disc are so blurred as to be indistinguishable; hæmorrhages and white patches in the retina may accompany this neuritis,

so that the whole ophthalmoscopic picture may resemble that of renal neuro-retinitis.

The neuritis is almost invariably bilateral, though I remember seeing a case of left cerebral abscess some years ago at Windsor, with Surgeon Colonel Harrison, of the Guards, in which the left eye appeared quite normal while the *right* presented well-marked optic neuritis.

General brain diseases rarely lead to ophthalmoscopic changes.

In at least half the cases of tubercular *meningitis* neuritis is a well-marked but late symptom; it is also commonly seen in traumatic and syphilitic affections of the meninges (Gowers).

Ocular signs and symptoms are common in *locomotor ataxy*. Of these one at least, **optic atrophy,** is of considerable importance, as it frequently precedes the general symptoms of the disease. Optic atrophy occurs in a large proportion, of all cases, 15 per cent. (Gowers) or 20 per cent. (Swanzy), and usually affects both eyes simultaneously. Very often the disturbance of vision produced by it is the first symptom for which tabetics seek advice, and I have known it precede all other symptoms by six months; it is a curious fact, too, that its presence is a favourable sign as regards the prognosis of the general disease, though, of course, the prognosis as regards vision is as bad as it can be. The ophthalmoscopic picture is a well-marked one: the disc is beautifully clear and sharply defined, grey or white, slightly cupped (edge seen with higher lens than floor), lamina cribosa lattice-work distinct; the vessels early become narrow, and grow narrower and narrower daily.

Treatment is quite useless; the defect of vision progresses steadily to complete blindness. The *Argyll-Robertson pupil* (reaction to accommodation but not to light) is so well known as a symptom of tabes that it need only be mentioned; it is frequently an early premonitory symptom. *Myosis*, or contraction of the pupil, often extreme, is present in most cases. Other eye symptoms found in tabes are paralysis of orbital muscles, epiphora, narrowing of the palpebral fissure, reduced tension, and imperfect closure of the eyelids.

The *Argyll-Robertson* pupil is often present in

congenital tabes (Friedreich's disease), but optic atrophy is very rare.

Ophthalmoscopic changes have very seldom been seen in cases of *cerebro-spinal meningitis*. Optic neuritis has been recorded in *myelitis*, but is of no diagnostic or prognostic value. In so-called "railway spine" functional eye symptoms are very common, but no eye lesions have been observed.

GENERAL DISEASES.

Tubercle of the chorioid is comparatively rare. It occurs either as small yellow rapidly growing patches symptomatic of acute miliary tuberculosis (prognosis, both general and local, bad), or as a single largish light-coloured tumour of chronic course, the prognosis of which is not nearly so bad, as there may be no other foci in the body. Early enucleation, in order to prevent the spread of the disease, is advisable in the latter form when diagnosis is certain.

The ocular changes in **syphilis** and **malaria** are very characteristic, and of special interest for Army surgeons; they will be discussed in later chapters.

Optic neuritis has been observed occasionally as a sequela of the **acute infectious fevers.** Retinal hæmorrhages are of frequent occurrence in **septicæmia** (almost invariably in puerperal septicæmia—Gowers, Litten); and metastatic panophthalmitis has been seen in **pyæmia.** The optic atrophy following some cases of facial **erysipelas** is probably due to extension of the disease to the optic nerve.

HEREDITARY TAINT—EXCLUSIVE OF SYPHILIS.

In certain families the male members are attacked about their twentieth year by optic neuritis (**Leber's disease**). Patients are often healthy in other respects, though usually of neurotic type; many in the recorded cases seem to have been excessive smokers of shag tobacco, and this appeared to act as excitant of the dormant tendency. This disease gradually goes on to atrophy, not however of a very well-marked form; central vision is greatly lowered, but the fields show

little tendency to contraction. In the few cases I have seen treatment was quite useless.

The remarkable disease known as **retinitis pigmentosa** is due to hereditary taint of an equally obscure nature. Congenital atrophy of the retina would be a better name, as it would include those cases in which the same signs, symptoms, and pathological changes are found, with the exception of the pigment. The disease is bilateral, and develops in childhood or early adult life. Both the subjective symptoms and the observed lesions are very characteristic. The disease should always be suspected when a young person complains of difficulty in seeing at night (night-blindness —hemeralopia) as this is one of the first symptoms ; this gradually increases till the patient has to be led about in the dark, though he has no difficulty in finding his way about in the daytime for a long time. On examining the field of vision it will be found concentrically contracted ; this contracting slowly increases, so that sooner or later day vision for practical purposes becomes *nil*, though central vision continues good. Finally, after many years, central vision is lost. On examining such a case with the ophthalmoscope a very peculiar atrophy of the retina is seen ; the whole fundus is greyish, the disc of a dirty yellow colour, the arteries and veins notably diminished in size ; most characteristic of all (but not invariably present), black branched spots, recalling at once the microscopic picture of bone corpuscles, speckle the retina, at first only at the periphery, but gradually encroaching back farther and farther till the macula and disc are reached ; in some cases the outermost zone of the fundus remains free from pigmentation.

Liebreich believed that parental consanguinity could be found in half the cases, but more recent observers have only detected this factor in about a quarter ; he also found that nearly 6 per cent. of deaf-mutes suffer from the disease. Idiocy is seen in from 12 to 13 per cent. (Hoering) ; congenital deformities are also frequent accompaniments, including colobomata of the various structures of the eye. It is stated to be fairly common in India. I believe the Jewish race is more predisposed

to it than others : of 21 cases seen by me at Moorfields, 15 were in Jews, but it must be remembered a very large proportion of Moorfields patients come from the London Ghetto. I have seen three cases in the Army—another proof, if proof be necessary, of the necessity of supplementing the "dot test" by ophthalmoscopic examination.

Cases of bilateral congenital amblyopia—apart from albinism—have been frequently recorded ; unilateral amblyopia is common enough. A few cases of congenital night-blindness without any pathological changes have also been reported.

TOXIC AMBLYOPIAS.

Of the various toxic amblyopias the only ones seen in military practice are those due to quinine (*vide infra*) and tobacco. **Tobacco amblyopia** is generally considered to be a chronic retro-bulbar neuritis ; recent investigations, however, tend to show that the nicotine poisoning probably first affects the retina, and ascends from there through the papillo-macular fibres of the optic nerve. It is always bilateral. The chief symptoms are (1) varying defect of visual acuity, (2) improved sight at night (nyctalopia), and (3) central scotoma for red and green. A cure—not, however, an early one—generally follows stoppage of tobacco. Ophthalmoscopic appearances are often *nil*, though sometimes marked pallor of the temporal half of the disc is present.

CHAPTER VII.

GONORRHŒAL DISEASES OF THE EYE.

If a disease be judged by the aggregate of misery and incapacity it entails, there is probably no greater scourge of the human race than gonorrhœa, not even excepting syphilis. When one realises the terrible fact that from one-third to one-half the total blindness of the world is due to gonorrhœal ophthalmia, the importance of the disease from the point of view not only of the ophthalmic surgeon but of humanity at large is manifest. Nor is ophthalmia the only gonorrhœal eye disease: gonorrhœal iritis is now recognised as a distinct entity; and the tendency of modern research is to show that other eye lesions may be due, wholly or in part, to the gonococcus.

GONORRHŒAL OPHTHALMIA.

Infantile gonorrhœal ophthalmia, or "ophthalmia neonatorum," is a violent form of purulent conjunctivitis, due to infection of the eyes as the child passes through the vagina of a mother suffering from gonorrhœa. Many will hold that this definition is too sweeping, and would add after the last word "or other purulent discharge"; and it is quite true that in a certain number of cases examination of the vaginal discharge of the mother fails to detect the gonococcus. It does not follow, however, that the gonococcus was not present *at the time of infection*, and apart from this consideration it is quite easy to overlook a microscopic organism in the complicated discharge from the vagina after pregnancy. On the whole, therefore, I think the above definition may be accepted with the reservation that some rare cases are due to other forms of purulent discharge; the more so as I am convinced it is of the utmost importance that the essentially gonorrhœal nature of "ophthalmia neonatorum" should be forcibly impressed on practitioners generally, and more especially

on Army surgeons in charge of the women and children of large garrisons. The expression "ophthalmia neonatorum" should be banished from medical nomenclature; it is misleading and dangerous, in that it is apt to engender, unconsciously perhaps, the belief that infantile gonorrhœal ophthalmia is a condition differing from the adult disease, not so grave in its immediate and after effects, and not calling for such prompt and energetic remedial measures—a very grave misconception.

Simple *catarrhal conjunctivitis* is common enough in infants, and should be carefully discriminated from the disease we are now considering.

Infantile gonorrhœal ophthalmia "is, with the single exception of atrophy of the optic nerve, the most frequent cause of blindness. The Committee of the British Ophthalmological Society reported in 1884 that from 30 to 41 per cent. of the inmates in four blind asylums in Great Britain owed their blindness to it. Reinhard, quoted by Magnus, found in 1876 that in twenty-two German blind asylums 658 cases out of a total of 2,165, or 30·5 per cent., were blind from this disease. The British Royal Commission on the Condition of the Blind estimated that there were about 7,000 persons in the United Kingdom who had lost their sight from this disease, or about 22 per cent. of the entire blind. Of the 50,568 blind persons in the United States, according to the census of 1890, Burnett estimates that at least 30 per cent. have become so from ophthalmia neonatorum." (Minis Hayes.)

From these considerations it is evident that there can be no greater boon to the individual and community than a simple and efficient means of *prophylaxis* against this fearful disease. Fortunately we have such means at our disposal, efficient beyond the reach of doubt; so efficient that a distinguished American ophthalmologist (Swan M. Burnett), in discussing the estimate that 15,000 of the blind in America owe their blindness to this disease, goes so far as to assert that "had proper measures been instituted at the right time not one of those 15,000 cases of blindness would have occurred." Glascott (Manchester Blind Asylum) estimates that nine-tenths of the cases of blindness from infantile gonorrhœal ophthalmia are preventible.

This method of prevention, known as Credé's method, from the distinguished Leipsic professor who first introduced it, is as follows :—

The attendant at a confinement cleanses the head, face, and eyes of the child, as soon as the head passes the vulva, with sterilised water or an antiseptic solution, then separates the eyelids and drops in from a glass rod one drop of a 2 per cent. solution of nitrate of silver into each eye. The inflammatory reaction is very seldom severe, and requires no special treatment.

The procedure is simplicity itself ; yet, simple though it be, it has reduced the percentage of infantile ophthalmia in the maternity hospitals in which it has been introduced from one varying between 19 and 4 to one of ·2, "and even this can usually be attributed to some neglect or carelessness on the part of the attendant" (Burnett). I have no hesitation in saying that any Army surgeon in charge of women and children is guilty of neglect if he omits to carry out, or cause to be carried out, Credé's method in all cases of confinement.

In Germany midwives are liable to punishment by the law if they do not report, or cause to be reported, all cases of sore eyes in newly born infants to the local authorities and the district doctor. In several States of America a similar law is in force, and it seems nothing less than a national sin of omission that some such legislation has not yet been enforced in England.

Adult gonorrhœal ophthalmia owes its genesis to precisely the same organism as infantile, viz. the gonococcus, introduced into the eye in the urethral discharge accidentally transferred from the genitals by the patient's hand ; hence it is supposed to be more frequent in the right eye of a right-handed, the left of a left-handed, man. Even a very slight, long-standing gleet is still dangerous, for so long as the gonococcus can be found in the discharge so long does the possibility of the inoculation of the eye with it persist.

The fact that adult gonorrhœal ophthalmia is relatively infrequent compared with infantile, and relatively rare as a complication of gonorrhœa, speaks volumes for the natural and acquired protective influence of the lids and lashes against the intrusion of foreign matter.

True, the face of the adult can never be actually bathed, and bathed for a considerable time, in gonorrhœal pus, as is the face of the infant; nevertheless, the minute percentage of gonorrhœal ophthalmia in persons suffering from gonorrhœa is a never-ceasing wonder when we consider the innumerable opportunities for accidental transference in the cleanliest of patients. I use the word "relatively" above advisedly, as the total number of cases of adult gonorrhœal ophthalmia in a community, such as an army, in which gonorrhœa is rife, forms a pretty formidable aggregate. It is, unhappily, a rare experience to visit a large military hospital without finding at least one case of gonorrhœal ophthalmia under treatment; although special precautions against the disease are taken in Army hospitals, such as the issue of venereal clothing (specially and separately washed), detailed warnings to the men, burning of dressings, etc.

The **prophylaxis** consists in warning all gonorrhœa patients of the danger; in directing them to cleanse their hands invariably after touching the genitals; in providing special underclothing, nightshirts, sheets, etc.; in burning all dressings; and in ordering all symptoms of eye irritation detected by patient or orderly to be *at once* reported. When a patient is seen *immediately* after the entrance of gonorrhœal discharge into the eye it is quite possible to avert the disease by prompt and energetic measures. I remember at Moorfields receiving a spirt of pus right into my left eye when incautiously separating the adhering eyelids of a child suffering from gonorrhœal ophthalmia. A colleague at once copiously irrigated the eye with 1 in 5,000 corrosive sublimate, and then instilled a 2 per cent. solution of nitrate of silver (which I am bound to say caused maddening pain for a few minutes), with the result that no ill effects followed. When one eye is already attacked, the other should invariably be protected by a bandage, shield, or other device after the fashion familiar to all hospital residents. Surgeons themselves are often inexcusably careless when attending such cases; the greatest care should be exercised in separating swollen adhering lids, as the pent-up matter often jets out

with great force, and protective goggles should always be worn when irrigating or dressing the eye.

Symptoms.—Symptoms may appear any time between six hours and three days from the introduction of the infectious material into the eye. The *first stage*, the length of which is in inverse proportion to the virulence of the attack, is heralded by itching and tickling and conjunctival injection ; soon the lids swell and become shiny, and a copious discharge of thin watery fluid containing whitish flakes appears. Even now it is not very easy to retract the swollen lids so as to inspect the globe ; when the globe is seen the conjunctiva is found much injected, with probably commencing chemosis (swelling of the conjunctiva at the corneal edge).

This stage rapidly passes into the *second*. The discharge becomes purulent and more copious ; the lids are enormously swollen and tense, the cilia matted together ; the eye can only be inspected with difficulty ; the conjunctiva is violently injected, and swollen so as to form a cushion round the cornea ; and the patient complains of considerable pain and heat in the eye and side of the face. In mild cases, and much more frequently in infants than in adults, the disease may be arrested at this stage, amelioration beginning after it has lasted a few hours or days ; and after a period varying from ten days to a month the patient may emerge with a weak, watery, swollen, but cured eye. Too often, however, the *third stage*, that of corneal disintegration, follows. The vitality of the corneal tissue is lowered—much earlier in adults than in infants—both from malnutrition, owing to pressure of the chemosis on its edge, and from being constantly soaked in pus ; particles of pus pocket beneath the overhanging folds of conjunctiva, the corneal epithelium "gives," and gonococci and pyogenic organisms enter, forming rapidly viscous yellow ulcers which will probably involve the whole, or nearly the whole, cornea ; then at the best a large leucoma is left, at the worst prolapse of the iris (or even exit of the lens) through a perforation or perforations. In some cases an actual necrosis of the whole cornea, from the pressure of the

ring of chemosis, seems to take place. The result in a prolapse case is a blind eye, the transparent cornea being replaced by an unsightly bluish white cone, in which membrane of Descamet, iris, and remnants of cornea are matted together ; and even in simple leucoma cases the corneal opacity is usually so large or so centrally situated that the small remaining amount of vision is useless.

Treatment.—Promptness, energy, thoroughness, and continuity must be the watchwords of the surgeon in treating this terrible disease, whether in infant or adult. Unless he can give his whole time to the case, which is unlikely, the surgeon must be aided by an experienced eye nurse, or better still by two nurses—one for the day, the other for the night. The first step is to protect the unaffected eye by means of Maurel's or Buller's shield, or a watch-glass in a ring of adhesive plaster ; if this be unavailable, the eye must be covered with boric lint, over which gutta-percha tissue or oiled silk is fixed by plaster and a bandage. In infants, unfortunately, the disease is generally bilateral when the patient is first seen, so that the necessity for this step does not arise. We now turn to the affected eye.

First Stage.—Many ophthalmic surgeons deprecate what they call "attempting to abort" the disease in this stage, and advocate simple irrigation with a mild antiseptic solution alone, without employing nitrate of silver. From my experience in the Army and the out-patient department of Moorfields, I am convinced this is a mistaken policy ; in nitrate of silver we have a germicidal agent of great power as well as an escharotic, and by its use in this stage we can most assuredly cut short—"abort" if you will—the vast majority of cases of infantile, and a certain number of cases of adult, gonorrhœal ophthalmia. On the appearance of any irritation of an eye in a case of gonorrhœa I always evert the lids, and brush the tarsal conjunctiva with a 5 or 10 grain solution, in addition to free irrigation ; and in doing this one is only, after all, carrying out the principle of Credé's method of prevention. If, as sometimes happens, the conjunctival irritation is only due to an accidentally coincident catarrhal con-

junctivitis, no harm is done; whereas if it be the first stage of gonorrhœal ophthalmia, the very best means have been taken for cutting short the disease, very probably in its second, just possibly in its first, stage.

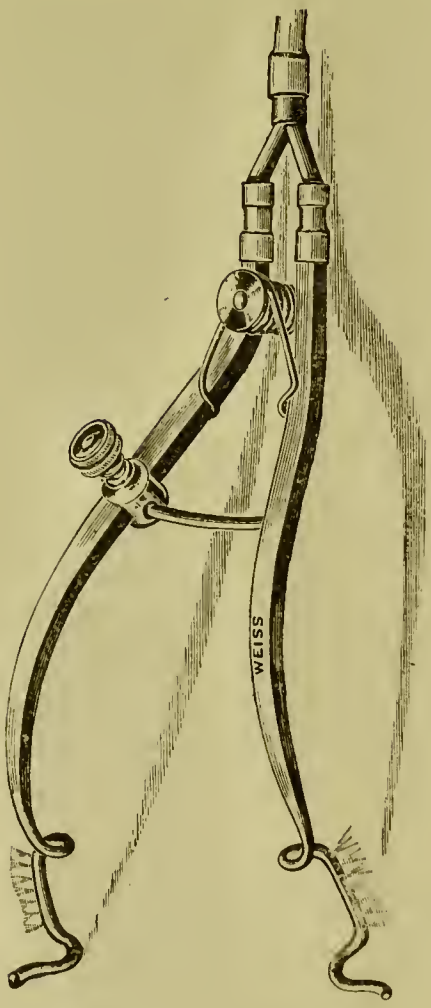


Fig. 18.—Hollow Speculum.

In the first stage, then, the treatment should include:—(1) Painting the everted lids with a 5 or 10 grain solution of arg. nit. once in twenty-four hours; neutralising by salt solution is quite unnecessary. I have tried other silver preparations, such as protargal and argentamine, but have not found them so reliable as the simple nitrate. (2) Thorough irrigation for two or three minutes four to six times daily with warm sterilised water or 1 in 5,000 corrosive sublimate. Longer or more frequent irrigations than this are inadvisable; the cornea is so quickly reduced in vitality in this disease, especially in the adult form, that

repeated or prolonged irrigation appears actually to wash it away. An *irrigator* should always be employed, never a syringe; an excellent way of employing it, which has the advantage of avoiding splashing and danger to the attendants, is to irrigate through a hollow eye speculum attached to the tube, or, better still, through the little bent tube invented by Dr. Ward Cousins. (3) The nurse should keep a piece of boric lint moistened with warm antiseptic solution over the eye in the intervals of active treatment, and exercise constant watchfulness to prevent the lids sticking together. Leeches are quite useless. In a case of adult gonorrhœal ophthalmia recently under my care I thought that benefit was derived from instilling extract of suprarenal capsule two or three times daily; certainly its immediate effect in blanching the conjunctiva was most marked.

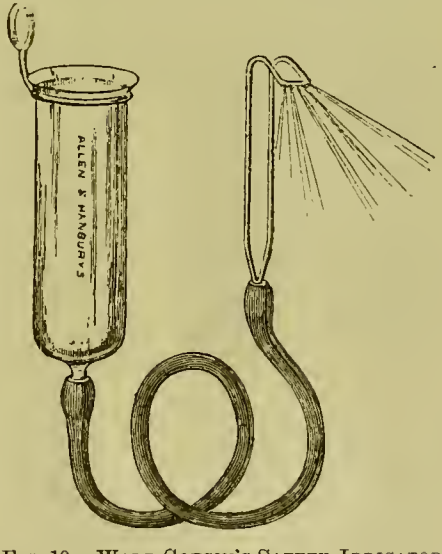


FIG. 19.—WARD COUSIN'S SAFETY IRRIGATOR.

The same treatment may be pursued in the *second stage* if the case be a mild one; but if severe the strength of the silver solution must be increased to 20 or even 30 grains to the ounce, and its application followed by brushing with salt solution, or the mitigated stick may be employed in its place. The eschar formed by the arg. nit. separates in from eight to twenty-four hours: the application should be made daily. Irrigation must be persevered in. Hot boric fomentations give great relief to the heat and pain, and seem of real utility; cold compresses or ice-bags are

inadvisable owing to the lowered vitality of the cornea. Many surgeons scarify the chemotic conjunctiva with a view to diminishing the pressure, but I have never seen any good result follow this procedure.

Special measures directed to the disease of the cornea may be added to the above in the *third stage*, such as instillation of atropine, iodoform, formalin, etc., but I fear they are of little service. Swan Burnett strongly deprecates the common practice of puncturing or snipping off prolapsed iris, in view of the danger of setting up panophthalmitis by the entrance of pyogenic organisms into the eye.

For the hypertrophy of the conjunctiva which persists for so long after the subsidence of the disease, I know of nothing better than daily light rubbing with sulphate of copper. A morsel of dilute yellow ointment inserted once or twice daily in the eye seems to help in dispersing corneal nebulæ. A blind and disfigured eye should be excised.

GONORRHŒAL CONJUNCTIVITIS.

A gonorrhœal conjunctivitis, due not to external infection, but to "the toxins of the gonococcus circulating in the blood," and occurring about the time arthritis is apt to set in, has been described. Such cases are, however, I believe, either (*a*) catarrhal conjunctivitis accidentally coincident, or (*b*) conjunctival injection preceding or accompanying gonorrhœal iritis or scleritis.

GONORRHŒAL IRITIS.

Iritis, or rather irido-cyclitis, is a much more common complication of the later stages of gonorrhœa than is generally supposed. Probably at least 10 per cent. of all cases of primary iritis are attributable to gonorrhœa; and recent investigations tend to show that many, if not most, of the cases of so-called "rheumatic" iritis are really gonorrhœal. Yeld[1] has analysed 159 cases of primary iritis; in 56 non-syphilitic cases, 28 (*i.e.* 50 per cent.) gave a history of gonorrhœa—of these 26 had a history of arthritis, past or present, and in 13 the

[1] Annual Meeting of British Medical Association, 1901.

association of gonorrhœa, arthritis, and iritis was so close as to justify the term "gonorrhœal" iritis.

It is almost always associated with gonorrhœal rheumatism, sometimes preceding, more generally accompanying, it; those who have had previous attacks of gonorrhœa and those with an inherited rheumatic tendency being most prone to it. In its most typical form it partakes of the characters of both plastic and serous iritis; there is general conjunctival congestion, from which, however, the violet peri-corneal ring of ciliary injection is easily distinguishable; the iris is immobile, has lost its sheen and become discoloured; early adhesions of the iris to the lens (posterior synechiæ) are so frequent as to be practically the rule; the aqueous looks faintly cloudy, like weak milk and water; and tiny white flakes of lymph, arranged in the form of a triangle apex upwards, can be detected by focal illumination on the back of the cornea (*keratitis punctata*). Pain is a distressing symptom, and always worse at night. Exacerbations of the eye affection often appear to alternate with the joint inflammations. The disease is generally unilateral.

The above is a pretty faithful description of a case I am at present attending—a young soldier suffering from gleet "off and on" for nine months, and now the victim of rheumatism of the right knee and ankle, and right iritis. The eye and the joints are not often acutely painful together; when the eye is bad the joints feel better, and *vice versâ*. His pupil under atropine has the exact shape of the ace of clubs, owing to posterior synechiæ; there are small dots of pigment on the lens, and very fine karatitis punctata; latterly some floating opacities in the vitreous have made their appearance, and he has had one or two attacks of increased tension.

In this form of iritis, as in others, atropine—four grains to the ounce instilled four times daily—is our sheet anchor, although it is very difficult to prevent synechiæ with it, even when the case is seen very early. The application of continuous warmth to the eye by means of a Japanese "hot box" is also most useful in relieving pain and hastening recovery; by its means *continuous* heat for several hours is ensured, and for

iritis it is certainly preferable to fomentations. With the eye treatment should be combined an energetic attempt to cure any urethral lesions remaining, and general anti-rheumatic treatment. A not infrequent complication is sudden increase of tension going on to a glaucomatous condition of the eye; for this a small iridectomy will be necessary, and will give immediate relief, but the outlook as regards useful vision in these cases is bad.

On the whole, it must be said the prognosis in gonorrhœal iritis is not a good one; atropine does not seem to act so quickly and beneficially as in other forms of inflammation of the iris; recovery without synechiæ is rare, with perfect vision rarer still; and, worst feature of all, relapses in the same or the other eye are more the rule than the exception.

Another form of gonorrhœal iritis—non-relapsing—has been described, but must be excessively rare. I have never seen a case. "It is not associated with any changes in the joints or fasciæ, and comes on during the earlier stages of gonorrhœa. It may affect one or both eyes, and be accompanied by symptoms of severe inflammation. As a rule there is free exudation, and sometimes the pupil is occupied by a considerable mass of bluish grey lymph. If the case be taken in hand early, complete cure usually comes about." (Brailey and Stephenson.)

GONORRHŒAL SCLERITIS AND SCLEROSING KERATITIS.

Both superficial and deep inflammation of the sclera (episcleritis and scleritis) are somewhat rare complications of gonorrhœa. I have only seen three cases that could justifiably be called gonorrhœal. In two of these a small circumscribed "nodule" or "boss" appeared in the sclera close to the cornea on its temporal side; in the third there were two such scleral bosses, one above, the other on the temporal side of, the cornea. These nodules were rounded, tender to the touch, and immovable, though the conjunctiva moved freely over them. The general conjunctiva was but slightly injected, and vision quite unaffected. Night pains were a marked

feature, and in one case very severe. In all three the scleritis came on at a late stage of the joint affection, and relapsed time after time; finally the men were invalided (for the joint affection), and I lost sight of them. In the intervals between exacerbations the affected patches of sclera were left slightly bulging, slatey coloured, and evidently thinned.

In the third case alluded to above little lunules of grey infiltration had pushed themselves out into the cornea from the bosses: a typical example of the "sclerosing keratitis" seen in gouty and rheumatic subjects.

The treatment of scleritis is most unsatisfactory. In one case I tried massage, but it only seemed to increase the pain without promoting subsidence. The usual anti-rheumatic remedies may be tried. The application of a "hot box" at night is of the greatest service in relieving the nocturnal pain.

RARE COMPLICATIONS.

Retinitis, neuro-retinitis, and bilateral inflammation of the lachrymal glands have been described among the ocular complications of gonorrhœa, but are of exceeding rarity.

CHAPTER VIII.

SYPHILITIC DISEASES OF THE EYE.

THE ocular manifestations of hereditary and acquired syphilis are numerous and important, and Army practice furnishes exceptional opportunities of studying this class of disease. Syphilitic eye diseases in the Army, more especially iritis and interstitial keratitis, are the source of considerable annual loss to the State, and from this point of view alone it is difficult to over-estimate their importance.

HEREDITARY SYPHILIS.

(*a*) **Interstitial Keratitis.**—We owe the elucidation of the intimate connection between this disease and hereditary syphilis to the brilliant researches of Mr. Jonathan Hutchinson, whose classical work on the subject has made the etiology and diagnosis of interstitial keratitis so well known to the profession in general. "Anterior uveitis" would be a more scientifically correct name for the disease, as the changes in the cornea affect those deeper layers which embryology shows to be part of the uvea, and the anterior portion of the uvea proper is always implicated; but the term "interstitial keratitis" has become a classic, and will probably always be retained as a distinctive name for that particular form of hereditary syphilitic disease in which the cornea is the part most obviously attacked.

It occurs in many, not in all, of the victims of hereditary syphilis between the ages of six and twenty-five, but most frequently about the eighteenth or nineteenth year; it is very common, and a cause of much inefficiency and invaliding among young soldiers in the Army. Patients suffering from it almost invariably present other signs of hereditary syphilis. Of these signs the most remarkable are the changes in the per-

manent teeth first described by Mr. Hutchinson, and generally associated with his name. "If the upper central incisors are dwarfed, too short and too narrow, and if they display a single central cleft in their free edge, then the diagnosis of syphilis is almost certain. If the cleft is present and the dwarfing absent, or if the peculiar form of dwarfing be present without any conspicuous cleft, the diagnosis may still be made with confidence. It is remarkable that the lateral incisors rarely show any peculiarity, but they also are sometimes dwarfed. The peculiarities in the other teeth (in the lower incisors, the canines, and the molars) are, so far as I know, of a kind which it is impossible to distinguish from those due to stomatitis." (Hutchinson.) Other signs are fine lines at the corners of the mouth, cicatrices of rhagades, enlarged glands in the neck, sinking of the bridge of the nose, ozæna, defective hearing, periosteal swellings on the tibiæ or other long bones. Army recruits presenting these evidences of hereditary syphilis should be unhesitatingly rejected.

"Interstitial keratitis in its typical form is always a consequence of syphilis, and is in itself sufficient for the diagnosis. It must, however, be carefully diagnosed. It usually begins by cloudiness of the substance of the cornea, with ciliary congestion and irritability. The clouds increase and coalesce till the whole cornea looks like ground glass" (Hutchinson). The infiltration, however, nearly always varies in density in different parts of the cornea, some parts of which may remain almost transparent. Professor Fuchs says that the formation of ulcers never takes place; this statement is, however, too sweeping. Undoubtedly ulcers are extremely rare epiphenomena, but cases do occur now and then in which the surface of very densely infiltrated spots breaks down and stains with fluorescin: I have seen two such cases at Moorfields, and others have been recorded by Hutchinson and Treacher Collins. The amount of vascularity varies astonishingly in different cases; in some no vessels can be detected even with a lens; in others—and these by no means the worst—the whole cornea becomes a dull pinkish-red, the so-called "salmon patch." In all cases there is more or less congestion and œdema

of the iris, and actual iritis with posterior synechiæ is not uncommon. The ciliary body visibly participates in the inflammatory process, frequently leaving a slatey discoloration of the sclerotic, similar to that following scleritis, on the subsidence of the disease. The changes in the chorioid are so far forward that even in cases in which part of the cornea remains clear they are seldom seen; but in the autopsies made they have always been present.

Both eyes are invariably affected, though one may precede the other by a period varying from a few weeks to many months. The disease runs a very chronic course, its duration being little, if at all, influenced by treatment. The opacity of the cornea reaches its acme within a few weeks, then very gradually the cornea clears, in many cases altogether, in many others sufficiently to leave useful vision; in a few, however, central nebulæ are left, and sight permanently impaired. I have frequently observed irregular astigmatism as a sequela. On the whole, prognosis is good, and is, I believe, better in vascular than in the non-vascular cases.

Treatment is useful in relieving symptoms, preventing complications, and in improving general nutrition, but I fear has no effect in shortening the course of the disease. Dark goggles must be constantly worn to relieve the photophobia, and prevent the troublesome sneezing on exposure to light which is a rather characteristic symptom. Atropine paralyses the iris, keeps the eye "quiet," and is of great service in obviating iritic complications. Warm applications, such as the Japanese charcoal box or hot fomentations, relieve the irritation and sometimes seem to hasten resolution; steaming the cornea, a procedure I have often seen carried out in Paris clinics, is also believed to accelerate the dispersion of the infiltration. As regards general treatment, the efficacy of mercury is very doubtful; on the whole, patients do best under a regimen of general tonics, such as cod-liver oil and iron, country air, and good food. Remaining opacities of the cornea, even of long standing, can often be cleared up in marvellous fashion by the long-continued use of dilute yellow ointment.

(*b*) **Chorio-retinitis.**—As noted above, the chorioid in the region of the ora serrata is always affected in interstitial keratitis; as a rule, however, the chorioidal lesions subside *pari passu* with the keratitis, or at all events cease activity on its subsidence. In some cases, however, fortunately not common, the keratitis seems to light up persistent disease in the chorioid and retina, which gradually spreads backwards from the periphery, and leads to serious damage to vision. The disease may present itself as a disseminated chorioiditis, with typical "worm-eaten" patches of atrophic chorioid surrounded by irregular black pigment; much more frequently, however, the whole periphery is covered with black speckles, which gradually spread backwards towards the macula, contracting the field and inducing night blindness, so that the condition often closely resembles retinitis pigmentosa. Another, fortunately rare, variety of hereditary chorio-retinitis is that in which, without any preliminary keratitis, yellowish patches of effusion appear in the macular region, leaving on subsidence large white areas of atrophy with much surrounding disturbance of pigment (central chorioiditis); such cases are seldom seen until the effusion stage is long past. Treatment is quite powerless to arrest the progress of these chorio-retinal lesions.

(*c*) **Infantile Iritis.**—This is apparently a rare complication of hereditary syphilis, but as it occurs at a very early age (usually between the second and eighth month) it doubtless often escapes notice. Of 23 cases summarised by Mr. Hutchinson, 12 were bilateral, 11 unilateral; in 15 there were copious effusions of lymph occluding the pupil, in the rest the iris was merely tumid and discoloured; in only 2 was any marked vascularity of the tunics noticed. As regards result, in 12 cases the pupil remained permanently occluded; in 8 cure seemed complete; in 3 the result was unknown. Infants suffering from iritis almost always show one or other of the recognised symptoms of hereditary syphilis; females are much more frequently affected than males, and the age of five months seems to be the period at or about which

the iritis is most likely to appear. Treatment, which is efficacious if early and energetic, consists of mercurial inunction and daily instillation of atropine. The efficacy of mercury in infantile iritis may possibly be connected with the fact that those who suffer from it are usually infants born within a short period of the primary lesion in the parents.

ACQUIRED SYPHILIS.

The most convenient way of studying the ocular complications, which are very numerous, of acquired syphilis seems to be to take the different parts of the eye and the syphilitic lesions thereof *seriatim*.

1. **Ocular Muscles : The Orbit.**—Paralysis of the ocular muscles are frequently, perhaps generally, due to syphilitic affections of the brain or cord (*vide* Chapter V.). The diagnosis and treatment of these diseases belong to the domain of general medicine. Tumours of the orbit and disease of its bony walls are often of syphilitic origin, and must be treated on general principles.

2. **Lids and Conjunctiva.**—The *primary sore* may occur on the skin of the lids, on their margins, or on the conjunctiva, and is often a puzzling mystery until its nature is suspected. In the cases I have seen the infection was conveyed by fingers which had been handling a sore ; a colleague of mine, when performing a circumcision, thus inoculated himself in the upper eyelid. The disease is said also to be frequently communicated by kissing. In most cases the characteristic induration at the base of the sore and the swelling of the pre-auricular and sub-maxillary glands help in clearing up the diagnosis ; but these features may be absent, and in one case under my own care (on the skin of the lower lid) I failed to make a diagnosis until a roseolar syphilide appeared. Care should be taken to eliminate all possibility of syphilis before diagnosing a sore as rodent ulcer. It is a good rule to suspect syphilitic inoculation or accidental vaccination in all cases of obscure sore of the eye or its adnexa. The treatment is the usual one, modified as regards local applications by the situation.

Tarsitis and *gummata* of the lids or conjunctiva are very rare, but cases of both have been recorded. Mucous patches may occur on the conjunctiva as on other mucous surfaces ; and the skin of the eyelids is, of course, liable to the various cutaneous eruptions.

3. **Cornea.**—Disease of the cornea in acquired syphilis is very rare ; this immunity of the cornea seems remarkable when we consider how frequently it is attacked in the hereditary form. Grey spots on the back of the cornea in the course of syphilitic irido-cyclitis are very frequent, and have been described as "syphilitic puuctate keratitis," but the term is a misnomer, as the lesions in these cases are only secondary to the irido-cyclitis, and partake of the nature of the keratitis punctata of serous iritis. Mr. Hutchinson describes an interesting case of true syphilitic keratitis in his work on syphilis. The keratitis, which was bilateral, and occurred simultaneously with the secondary eruption and the sore throat, assumed a most peculiar symmetrical form. The centre of each cornea was hazy, and towards it greyish striæ, each containing a minute blood-vessel, converged from the periphery ; both haze and striæ were distinctly in the layers of the cornea. The opacities remained almost unchanged for over twelve months, and then slowly cleared, leaving almost perfect vision ; the patient was more or less under mercury the whole time. In most other recorded cases the relationship between the keratitis and the syphilis is by no means clearly made out. Diseases of the cornea are common, and syphilitics are just as liable to suffer from them as others, but in the vast majority of cases the association between the disease processes is accidental. Corneal ulcers occurring in syphilitics do not appear to be affected in any way by mercury administered for the constitutional disease.

4. **The Iris and Ciliary Body.** — Syphilis accounts for fully one-half of the total number of cases of iritis, which is one of the commonest manifestations of the earliest secondary stage of the disease, and may occur at much later periods. Four distinct varieties of syphilitic iritis are met with. Of these, the first two are associated with the period of secondary eruption,

while the third and fourth are not seen till many years after the primary sore.

(*a*) *Plastic Iritis.*—This usually makes its appearance between the fourth and eighth months succeeding the primary infection. In a typical case the patient presents himself complaining of pain, photophobia, and lachrymation; the iris is immobile, discoloured, and the pupil probably already deformed owing to posterior synechiæ; the aqueous is diminished, and there may be a few spots of keratitis punctata due to lymph thrown up from it. On instilling atropine large synechiæ will be made evident, while smaller ones will be probably torn away, leaving pigment spots on the lens; the synechiæ are usually much broader than those seen in gonorrhœal or rheumatic iritis, but this in itself can hardly be looked on as a distinctive feature.

From this description it will be seen that there is nothing in the iritis itself to distinguish the disease with certainty from non-specific forms; the diagnosis of syphilis as the cause of this form must be purely inferential from the presence of other symptoms or from the history.

Occasionally the discomfort is so slight that the patient does not consult the surgeon until the activity of the disease process is over. Yet in this class of case —so-called "quiet" iritis—the resulting damage to the eye from synechiæ, pigment spots on the lens, and floating opacities in the vitreous, which the patient sees as annoying black specks, is often serious.

I have seen a very large number of cases of syphilitic iritis in the course of my service, and have found this plastic form much the more common variety.

(*b*) *Papular Iritis.*—Many ophthalmologists write as though this were practically the only form of iritis observed in secondary syphilis. Mr. Brailey goes so far as to say: "We may accept them (the papules) as occurring at some stage in the course of every case of specific iritis." This, however, is not my experience, nor is it that of most ophthalmic surgeons whom I have questioned on the subject. Undoubtedly papules are often present, but they are much more often absent, or at all events not discernible; and the fact that papular

iritis is brought forward so prominently in most text-books, while other varieties are barely, if at all, mentioned, leads beginners to make frequent blunders in diagnosis.

Papular iritis is also an early secondary manifestation; in fact it is simply plastic iritis *plus* nodules or papules. These papules are very small, seldom more than three or four in number, embedded in the iris, generally at the pupillary margin, sometimes at the periphery, very rarely indeed in the intervening portion; they never appear in any non-specific form of iritis, and their presence in a given case renders the diagnosis of syphilis a certainty. Beginning as round reddish points, they grow lighter from day to day, and finally either disappear or, if at the edge of the pupil, adhere to the lens and form very strong broad synechiæ.

Both plastic and papular iritis are unilateral, in the sense that only one eye at a time is attacked; but sooner or later the other eye is very liable to develop the disease. In both the ciliary body is affected, and occasionally the whole uveal tract participates, leading to opacities of the vitreous; but as a rule cyclitis is not a prominent feature. In the third variety, which I am now about to describe, the ciliary body suffers more than the iris.

(*c*) *Tertiary Irido-cyclitis.*—This variety of specific iritis is met with only in the so-called tertiary stage of syphilis, many years (generally ten to fifteen) after the primary sore. Fortunately, it is not very common, for it is exceedingly intractable, and almost always causes serious impairment of vision. In this form synechiæ are unusual, the iris being merely dulled or slightly discoloured; on the other hand, however, there is considerable turbidity of the aqueous, well-marked keratitis punctata, and frequently vitreous opacities, indicating serious affection of the ciliary body and uveal tract. Increased tension and secondary glaucoma are exceedingly apt to occur, owing to blocking of the filtration angle by lymph or by the tumified iris. Tertiary irido-cyclitis usually attacks both eyes simultaneously or almost simultaneously, but I have seen cases in which one eye alone was affected. In the absence of history or evidence of syphilis, diagnosis can only be inferential.

(*d*) *Gummatous Iritis.*—I have never seen a case of true gumma in the iris or ciliary body, but instances have been recorded. Gummata may grow to such a size as to burst the globe, but in some cases have proved very amenable to treatment, melting away rapidly under mercurial inunction.

Prognosis is grave in the tertiary and gummatous forms of iritis, mainly owing to the frequent onset of secondary glaucoma; even in comparatively mild cases the transparency of the media is liable to be permanently reduced by the keratitis punctata and vitreous opacities. In plastic and papular iritis complete recovery, *i.e.* recovery without synechiæ, cannot often be hoped for; synechiæ are the rule in early specific iritis, a fact on which sufficient stress is hardly laid in books. On the whole, however, prognosis is good as regards recovery of useful vision when treatment has been undertaken sufficiently early in the case to permit of incipient synechiæ being torn through by the atropine. Relapses are not so common as in gonorrhœal or rheumatic cases.

Treatment may be summed up in three words: atropine, heat, and mercury. The main point is to dilate the pupil *early*, and to keep it dilated. The atropine, in four grains to the ounce solution, should be instilled four times daily; heat is best applied by means of a Japanese hot box, wrapped in lint bandaged over the eye. Mercury in early secondary cases should be pushed to salivation as speedily as possible, and then administered so as to keep the gums lightly touched throughout the disease; in tertiary cases a prolonged course of the iodides of mercury and potassium is sometimes singularly efficacious. Increased tension calls for an early iridectomy; the operation is not always easy to perform, however, owing to synechiæ, and the result is sometimes very disappointing. In the case of complete annular synechiæ the operation should be performed at once, in order to restore communication between the chambers. For vitreous opacities, which are often of considerable size, a prolonged course of iodide of potassium is the best treatment.

5. **The Chorioid and Retina.**—(*a*) *Syphilitic*

chorio-retinitis is generally seen within the first two years after infection; it may come on insidiously without previous eye affection, but as a rule is preceded by iritis or irido-cyclitis. It is usually advanced when first seen, especially when unilateral, as for a long time its effect on vision is slight.

Patients generally present themselves complaining of difficulty in finding their way about at night, and of blurred vision during the day; the fields will probably be somewhat contracted, but not nearly so much as might be expected; scotomata are frequent. In a typical case the ophthalmoscopic appearances are pathognomonic. The whole fundus looks pale and "smoky"—the latter appearance being mainly due to a fine vitreous haze—and is dotted all over with fine black spots as though peppered; peripherally the peppering is thick, and many of the spots show a tendency to coalesce, while towards the posterior pole they are sparse and discrete; the disc looks blurred, and, contrary to the rest of the fundus, pinker than usual. In another and grosser form of the disease the picture closely resembles that of disseminated chorioiditis, and there is no doubt that some cases of disseminated chorioiditis are due to acquired syphilis; oval white atrophic patches are scattered over the fundus, more especially at the periphery, where they are very apt to be grouped in threes; the pigment disturbance round these patches is usually slight and much more regular than in disseminated chorioiditis; the ringed patches of atrophy are often beautifully regular and symmetrical. Most frequently, perhaps, both these forms occur together. Yet another form, in which the retina is mainly, if not entirely, implicated, is indistinguishable from retinitis pigmentosa, and may, I think, be justifiably called "acquired retinitis pigmentosa."

In all forms the phenomenon most characteristic of syphilis is the *fine* vitreous haze. It is so fine that it is easily overlooked by the ordinary methods of examination; it is best seen by the direct method, with feeble illumination and the small *plane* mirror.

The progress of the disease is slow and insidious, and according to my own experience prognosis is always

grave, though I have never seen a case in which vision was entirely lost. Treatment consists of mercury, preferably by inunction; dark glasses should be worn, but the practice of confining patients for long periods in a dark room is a barbarous one, the *raison d'être* of which it is difficult to see.

Among very rare chorio-retinal complications of syphilis may be mentioned the "tumid chorio-retinitis" of Hirschberg, and the "relapsing central syphilitic retinitis" first described by Von Graefe. I have never seen a case of either.

(*b*) *Syphilitic arteritis* in the retina is fortunately uncommon, as it nearly always leads, sooner or later, to hæmorrhages and blindness. It is a very late manifestation of the disease. The fundus picture is a curious one. Many of the vessels appear quite normal, while others are converted into thick white lines, within which close observation may detect a thin column of blood. After a time one or more of these white arteries will break down, covering the area of its distribution with hæmorrhages, often of large size. A beautiful picture of this condition will be found in Haab's Atlas, Fig. 28. *Syphilitic hæmorrhagic retinitis*—excluding cases of chorio-retinitis with minute punctiform hæmorrhages—without evidences of arteritis, as above, is exceedingly rare. I have quite recently had a young soldier under my care for sudden loss of vision in the right eye. Examination showed several small fresh-looking hæmorrhages in the macular region; the iris of the same eye was adherent to the lens in two places; vision = fingers at two feet. His "medical history sheet" showed two previous entries, one for primary syphilis two years before, the second for iritis nine months ago. Urine was quite normal; heart normal; no history of rheumatism. He had been an excellent rifle shot—a "marksman"—after the attack of iritis, and always shot from the right shoulder. The other eye was quite normal, and there was no error of refraction in either.

6. **The Vitreous and Lens.**—*Vitreous opacities* of all sizes and shapes, from fine specks to flakes, streaks, "spider's webs," or huge irregular bodies con-

cealing the fundus, are common in syphilis as the sequelæ of irido-cyclitis or chorio-retinitis. They are generally floating, and cause much disturbance of vision and annoyance to the patient. When of any size they are easily visible in the dilated pupil by transmitted light, when they appear as black moving objects on a red ground; a good method of observing them is to get the patient to look up and down rapidly, and then straight into the mirror. A more minute examination can be made by the direct method, with the plane mirror and feeble illumination in the case of very fine specks (*vide supra*). Cases of vitreous opacities without visible fundus lesions are not infrequent, but in such cases the lesions are probably too far forward to be seen. The treatment is the treatment of the cause.

There is no syphilitic disease of the *lens per se*, but stellate opacities due to deposit on its posterior surface are occasionally seen in cases of advanced chorio-retinal disease; such opacities are frequently seen in pigmentary retinitis.

7. **The Sclerotic.**—*Gummata* of the sclera have been observed, but are excessively rare. Many cases of scleritis and episcleritis give a history of syphilis, but the connection is very doubtful.

8. **Optic Nerve.**—Syphilitic optic atrophy is generally the herald of locomotor ataxy, though cases without ataxic symptoms are occasionally seen.

I have omitted allusion to lachrymal affections, which are often due to hereditary and acquired syphilis, in this chapter, as they will be more conveniently discussed later (*vide* Operations).

CHAPTER IX.

MALARIAL AFFECTIONS OF THE EYE.—QUININE AMAUROSIS.

TEXT-BOOKS on diseases of the eye, as a rule, dismiss the subject of malarial eye affections in a few words; in fact, I am only aware of one[1] in which anything like an adequate summary of the state of present knowledge of this important class of disease is given. When we consider the large amount of space often devoted in these works to diseases and congenital defects of excessive rarity—to the curiosities of ophthalmology, so to speak—the absence of detailed reference to malaria becomes astounding. On the other hand, a few able monographs on the subject are in existence, notably one by Sulzer; and a mass of evidence in the shape of articles on symptoms, notes of cases, etc., may be laboriously disinterred from the ophthalmological literature of the last thirty years. I append footnotes giving references to this and the following chapters, in the hope that they may prove useful to fellow workers in tropical ophthalmology.

Most medical men in practice in the tropics are familiar with a distinctively malarial class of eye diseases, with characteristic symptoms, characteristic ophthalmoscopic signs, and, above all, characteristic pathology; and few writers on tropical diseases omit more or less detailed allusions to the subject—indeed, so far back as 1833 we find a special "amaurotic" form of malarial fever described[2]—but only too often the value of their remarks is minimised by vagueness of

[1] "Traité Complet d'Ophtalmogie," by L. de Weeker and G. Landolt (article by J. P. Nuel).

[2] Stosch: "Febris Intermittens larvata amaurotica," *Carper's Wochenschrift*, 1833, 11, No. 3.

description and unscientific phraseology, those convenient but antiquated terms "amaurosis" and "amblyopia" being employed to cover a multitude of sins of omission and inaccuracy. Some idea of the importance of these affections, as well as of their widespread prevalence, may be gathered when we find it stated by Poncet[1]—to whose able and laborious researches we owe much of our knowledge on the subject—that he found fundus lesions visible by the ophthalmoscope in no less than 10 per cent. of malarials examined by him in Algiers, and by the microscope found fundus lesions *the rule* in necropsies on cases of malarial cachexia and pernicious malaria. It is therefore evident that malaria—which, like syphilis, spares no organ—does not spare the eye. In the brief space at my disposal I can only endeavour to bring together, in the form of a concise summary, facts hitherto to be found only in scattered form, supplemented by such observations as my personal experience in the tropics, at Netley, and at Moorfields enable me to make.

I purposely postpone reference to diseases of the anterior half of the eye to the latter part of the chapter.

Malarial eye lesions may be conveniently classified under the following heads :—

(1) Neuritis.
(2) Retinal hæmorrhages.
(3) Retino-chorioiditis.
(4) Effusions into the vitreous.

(1) Malarial Neuritis.

From one of my Hong Kong case-books I take the following brief notes of a typical case of malarial neuritis :—

Private A. F. admitted to hospital complaining of supra-orbital pains, dimness of vision, and photophobia.

During preceding twelve months had been in hospital seven times with malarial fever, no attack of exceptional severity ; he is thin, anæmic, spleen slightly enlarged ; temperature normal, urine normal. Vision $\frac{6}{60}$ each eye ; fields normal ; colour perception unimpaired ; disc raised, reddish grey, margins blurred ;

[1] *Ann. d'Oc*, 1878, t. lxxviii., p. 201.

rest of visible fundus normal save for a slight haze. He was placed on light, nutritious diet—chicken, fish, beef-tea, and given quinine. A week later the visual acuity had improved to $\frac{6}{18}$, but reverted after three days to $\frac{6}{60}$, and varied between that and $\frac{6}{24}$ for some time; five weeks after admission it suddenly became $\frac{6}{12}$, at which it remained. The supra-orbital pain and photophobia ceased a fortnight after admission. After seven weeks in hospital, he was sent to the sanatorium on the "Peak," and from thence invalided to England a month later with "malarial cachexia." Examined again before leaving, visual acuity $\frac{6}{12}$ fields normal, with the exception of a very slight contraction on the nasal side, colour perception normal; fundus normal, with the exception of a slight greyness on the temporal half of optic disc.

Signs and symptoms binocular throughout. No history of syphilis.

I have selected the above case as embodying most of the characteristics of the disease.

(1) It will be seen that the patient had suffered from repeated attacks of malarial fever. This is the case as a rule; it almost invariably occurs in those whose systems are broken down by many—it may be slight—attacks of fever. I have seen one case of neuritis commencing in the course of a pernicious attack, but such cases are not common.

(2) In the commencement supra-orbital pain and photophobia are almost constantly present, night blindness frequently.

(3) Colour perception remains unimpaired, except in the rare cases ending in complete atrophy.

(4) The variations in the visual acuity in the course of the malady form the most characteristic symptom, and distinguish malarial neuritis from all other forms. Sulzer, in an admirable paper on the subject,[1] to which I am largely indebted, lays stress on this point. A diminution of the visual acuity to one-tenth can rise in

[1] "Troubles de la Vision dans l'Impaludisme," *Arch. d'Ophtal.*, 1890.

two or three weeks to one-half or one-third, falling again perhaps in two or three days. Macnamara[1] described a case in the Calcutta General Hospital of a young girl of thirteen, admitted with a visual acuity perception of light only, who was discharged five weeks later with normal vision.

(5) Fields intact, or only slightly contracted.

(6) The fundus changes visible with the ophthalmoscope include swelling of the papilla, which assumes a greyish red colour, œdema of the circum-papillary retina, with effacement of the papillary margins and enlarged and tortuous veins. The peculiar coloration of the papilla—"teinte rouge-grisatre"—due to parasites in its capillaries, is pathognomic. In about a third of the cases tiny peripheral retinal hæmorrages are also found.

(7) About 80 per cent. of cases terminate in a partial atrophy, indicated by varying diminution of visual acuity, irregular contraction of the field, and slight greyness of the disc; many end in apparently complete recovery, some rare cases go on to complete atrophy.

Pathology and Morbid Anatomy.—For our knowledge of this branch of the subject we are mainly, if not entirely, indebted to Poncet,[2] who systematically examined with the microscope the eyes in all cases of death from malaria at the military hospital of Philippeville (Algiers). His researches show conclusively that the changes in the disc and retina in this disease are due primarily to melanæmia with increased vascularisation, the subsequent atrophy or partial atrophy being explained by consecutive endo-arteritis of the vessels. A perpendicular section through the retina shows the raised, swollen papilla (*tête de hanneton*), its little capillaries stuffed with leucocytes, each containing a central spot of black pigment—red corpuscles excessively rare; the same pigmented leucocytes (when Poncet uses the word "leucocytes" he undoubtedly means, in most instances, malarial parasites) fill the retinal vessels, and when hæmorrhages exist they are found to be due to

[1] *Medical Times and Gazette*, May 2nd, 1868.

[2] *Vide supra.* Also "Atlas des Maladies Profonder de l'Œil," by Perrin and Poncet.

emboli of these leucocytes with consecutive extravasations.

The affection is always binocular, although it does not usually begin in both eyes at the same time.

Sulzer believes that a certain proportion of these cases have malaria as a predisposing cause only, the exciting cause being the indirect action of sunlight, and adduces certain cases seen by him in Borneo in support of his view.

(2) Retinal Hæmorrhages.

Two varieties of retinal apoplexy are found in association with malaria: (*a*) minute peripheral, (*b*) large peri-papillary and macular.

Minute hæmorrhages in the ciliary zone of the retina are frequent in acute attacks of fever; they are often so very minute and so far forward as to be easily overlooked. Poncet found them in all cases of death from malaria. They may accompany or follow neuritis,[1] but often form the only apparent lesion of the fundus. It seems probable that many of the transient disturbances of vision so commonly seen in malarial fevers are due to slight œdema of the retina, followed by these tiny hæmorrhages.

The large peri-papillary and macular hæmorrhages are much less frequent, and, like the neuritis which they sometimes accompany, are usually seen only in malarial cachectics. These are of much graver import, always causing some impairment of vision, and occasionally even absolute loss. I believe some of the cases of "sudden and persistent amaurosis," described by writers on malaria to be due to macular hæmorrhages. The only case of such sudden amaurosis I have seen was in Hong Kong, in a soldier suffering from advanced malarial cachexia, who subsequently died; in the course of one night his vision in the right eye was reduced to perception of light, in the left to $\frac{6}{36}$; examination showed a large macular hæmorrhage in the right, with several

[1] Guenceau de Mussy: *Journal d'Ophtal.*, t. i., p. 5, 1872.

small hæmorrhages between papilla and macula in the left ; diffuse haziness of retina in both.

In a case of malarial cachexia invalided from the Indian frontier, which I recently saw at Netley, by the courtesy of the Director-General, Army Medical Staff, there were several hæmorrhages arranged in a curiously symmetrical manner along the inferior temporal vessels in both eyes ; V.A. in R. $\frac{6}{12}$, in L. $\frac{6}{24}$; urine normal.

Microscopic examination shows these retinal apoplexies in malaria to be due to infarcts of parasites, followed by extravasations (*vide supra*).

(3) **Retino-chorioiditis.**

In about 20 per cent. of acute intermittents, generally towards the end of the hot stage, patients complain of supra-orbital pains, tenderness on pressing the eyeballs, photopsies, and photophobia. Examination then discloses a general hyperæmia of the fundus, mainly venous ; red slightly swollen papilla, surrounded by a grey veil ; and general haziness of the retina, which appears to have an undulating surface—"dunes" with intervening depressions.

This œdematous state of the ocular membranes—the first stage of malarial retino-chorioiditis—generally subsides without leaving any apprcciable trace. In a certain number of cases, however, more especially in those who have had repeated attacks of fever and are falling into the condition of malarial cachexia, the symptoms persist, punctate peripheral hæmorrhages appear, and a chronic, slowly progressive retino-chorioditis is set up, ending in capillary atrophy of the choroid and much loss of vision. Out of the 38 cases of malarial cachexis which I examined at Netley in April, 1899, I found this condition of capillary atrophy of the choriod in three—nearly 8 per cent. I was able to watch the progress of such a case in a man—a discharged soldier who had suffered much from malaria in Burmah — who attended Moorfields for the twelve months. The fundus is now of an almost uniformly grey colour, as though powdered over with pepper, the disc is pale, and the arteries

reduced to fine threads ; the distribution of the chorioidal vessels is mapped out with extraordinary clearness, the vessels seem almost white, with a central red streak ; pigment-layer of retina and chorio-capillaries atrophied: vision $\frac{4}{60}$ in one eye, $\frac{6}{60}$ in the other ; some myopic astigmatism, but the correcting glasses only improve to $\frac{6}{36}$ and $\frac{6}{18}$; fields irregularly contracted. When first seen he had only general haziness and loss of lustre of retina, with V. $\frac{6}{12}$ in each eye with correction.

The following description by Poncet[1] of the retina of an Algerian colonist who died from a pernicious attack of malarial fever shows clearly the nature of the circulatory changes in the acute stage of this affection. The patient was much emaciated, pale, and anæmic, and had had several attacks of fever.

"In all the capillaries such a quantity of pigmented elements is found that each vessel looks as though formed of a mosaic with little black points. Red corpuscles are rare. Each little black point is a pigment molecule in the protoplasm of a white corpuscle ; very rarely the pigment is free in the capillary. Two points are very clearly demonstrated. First, the extraordinary number of leucocytes ; second, the enormous quantity of pigment. It is easy to understand the obstacle to the circulation caused by this mass of leucocytes in the capillaries. This abundance of pigmented leucocytes (? parasites) is found in the entire retina, at the periphery as at the papilla, and explains the peri-papillary œdema and dirty grey aspect of the papilla in these cases of malarial cachexia."

(4.) Effusions into Vitreous.

White Infiltration of Vitreous.—This rare and curious affection was first described by Seely,[2] and consists

[1] Planche, lix., Fig. 1. Atlas de Perrin et Poncet.

[2] W. W. Seely : "Transactions of American Ophthalmological Society Annual Meeting, 1882," p. 345.

in an infiltration of the vitreous, forming in stages, causing almost complete loss of vision for a time, and giving a characteristic white reflex with reflected light. Seely attributes it to a serous infiltration due to chronic paludism. In his two cases the progress of the disease was oscillating for several months; eventually the visual acuity became normal under prolonged quinine; in one case mobile opacities persisted.

Sulzer describes three cases of this disease. In the first two the ocular affection had existed some weeks before they came under his notice. In the first the eyes had been blinded during the night, with only a day's interval between each; in the second there was an interval of several weeks. The first was complicated by intense ciliary and supra-orbital neuralgia on both sides, with tenderness of the eyeballs and limited and painful motility. Both cases were malarial cachectics, with intense anæmia. Visual acuity was reduced to perception of light. The diffused vitreous infiltration, which at first gave a perfectly white reflex in all directions, was not completely absorbed in either case. During the eighteen months under observation this was replaced by moving flocculi arranged like a spider's web, with slight turbidity of the vitreous in the meshes, so that the fundus was only indistinctly visible, although the white atrophic colour of the papillæ could be made out. The right eye of the first case became blind, the left counted fingers at a short distance. The second counted fingers at 60 c.m. when last seen. The third case was seen a few days after the onset of the disease, when only the right eye was attacked; it presented a diffuse and equal infiltration of the posterior segment of the vitreous, with a faint peripheral red reflex on complete dilatation of the pupil; V.A. = fingers at 20 c.m. Four weeks later the left eye was similarly attacked. In three months, under quinine, total re-absorption took place, leaving V.A. normal.

Penoff[1] also described diffuse opacities of the vitreous in malarials, but they were complicated by affections of the uveal tract.

[1] *Centralblatt für prakt. Augen.*, 1879, p. 80.

I have never seen a case of white infiltration, and such cases of vitreous opacities in malarials as I have seen have been preceded or accompanied by uveal or other lesions only indirectly due to malaria.

It will, I hope, be clearly understood from the foregoing necessarily imperfect sketch that the classification I have attempted to make is based only on the prominence of one set of signs or another in the majority of cases. For instance, a sharp line of demarcation cannot always be drawn between neuritis, retinal hæmorrhages, and retino-chorioiditis ; many cases occur in which all three affections are associated or form stages in one process.

In the class of cases I now propose to deal with—mainly diseases of the conjunctiva, cornea, and iris—the evidence of malarial causation is not so satisfactory as it is in the case of malarial affections of the fundus, as the demonstration of the plasmodia in the affected tissues is generally lacking. Nevertheless, in many of these cases the suggestion of malarial origin is at least plausible, and in some the clinical evidence in favour of paludism being the *causa causans* appears conclusive. A brief summary of the salient features of these diseases, with references to the now recorded cases, may therefore be of service to those interested in tropical eye diseases, and will complete the survey of the subject.

Conjunctivitis.

At least three different varieties of conjunctivitis have been found connected with malaria, namely, (*a*) intermittent ophthalmia, (*b*) conjunctival injection due to neuralgia of fifth nerve, (*c*) epidemic conjunctivitis.

(*a*) *Intermittent ophthalmia* is thus described by Griesinger[1]: "It is nearly always unilateral, and consists of a more or less marked hyperæmia of the eye, with photophobia, lachrymation, contracted pupil, and frequently swelling of the lids." There is some discomfort, but no neuralgic pain ; the symptoms either come on during the attacks of fever in acute intermittents, or

[1] Quoted by Raynault, "Troubles Oculaires de la Malaria." Paris, 1892.

replace the paroxysm altogether; in the intermissions the eye is quite healthy: ordinary treatment fails to cure, but it yields readily enough to quinine. Curiously enough, the left eye is nearly always the one affected. That this form of conjunctivitis accompanying or replacing the paroxysms of acute intermittents is a real entity is undoubted in view of the many cases collected by acute and cautious observers; that it is directly due to malaria appears almost equally certain. Morton, as far back as 1727, described a case of tertian fever, apparently cured by quinine, which recurred later in the form of a tertian ophthalmia. Strack, in his work on intermittent fever published in 1785, gave notes of a case of quotidian congestion of the left eye, without pain or fever, in a malarious patient. Beylot saw several cases of the disease in the course of an epidemic of malarial fever at Biskra, in Algeria; and Raynaud[1] has published notes of three cases which he watched carefully, and in which the connection with malaria seems fully established. De Schweinitz[2] also, whose authority none can question, states that he has seen instances where the ordinary manifestations of malarial fever were replaced by this intermittent ophthalmia. Many other observations will be found quoted in Raynaud's excellent work. Most of the published cases were observed in Algeria and the United States; the disease must be very rare, if not unknown, in the Far East, as I never saw an instance in the course of four years' residence in China.[3]

(*b*) *Conjunctival injection due to neuralgia of the fifth nerve* is very common in malaria and malarial cachectics, as every practitioner in the tropics is aware, but can only be considered indirectly due to malaria. Pain in such cases is severe, and the conjunctival injection comparatively slight.

(*c*) *Epidemic Conjunctivitis.*—Epidemics of conjunctivitis have been attributed to malaria, notably that in South Carolina in the summer and autumn of 1882.

[1] *Op. cit.*
[2] *Medical News*, Philadelphia, June 7th, 1890.
[3] Yarr: *Journal of Tropical Medicine*, April, 1899.

The concurrence of the disease, however, is probably accidental.

Xerosis.—An affinity has also been traced between epithelial xerosis and malaria, and a connection undoubtedly exists, but probably only inasmuch as xerosis is the local expression of a general malnutrition of which malaria may be one cause.

Keratitis.

The various forms of keratitis which have been described as malarial may be conveniently grouped under three heads, (*a*) dendritic keratitis, (*b*) keratitis profunda, (*c*) vesicular keratitis (herpes corneæ).

(*a*) *Dendritic Keratitis.*—Kipp (Newark, U.S.A.) has made a very careful study of malarial keratitis, and there seems little doubt that the "dendritic" form described by him, Van Millingen, and others is not merely associated with, but directly due to, malaria. Kipp's first description of the disease appeared in 1880; in 1889 he published his most recent results.[1] In all he has observed this peculiar form of keratitis in no fewer than 120 malarial patients, in whom the connection between the malarial and corneal affection appeared to be quite clearly established. Beginning, in the course of or after a paroxysm of fever, with photophobia, lachrymation, and supra-orbital neuralgia, the characteristic lesion of the cornea soon made its appearance, a peculiar narrow serpiginous superficial ulcer with lateral offshoots, like the skeleton of veins in a lanceolate leaf. Anti-malarial treatment cured most cases rapidly; in a few, however, it had to be supplemented by local applications where the ulcerative process tended to penetrate deeply into the cornea. Kipp's observations were soon confirmed by several other American surgeons, notably Hotz, Miller, Sutphen, and Noyes; the last named drew special attention to anæsthesia of the cornea and exaggerated tenderness of the supra-orbital nerves as characteristic symptoms of the disease. The malarial dendritic keratitis described by Van Millingen in 1888[2]

[1] "Transactions of American Ophthalmological Society," 1889.
[2] *Centralbl. f. prakt. Augenheilk*, Jan., 1888.

is evidently the same disease, "fungus-like lesions, with ciliary neuralgia and anæsthesia of the cornea."

A valuable paper, entitled "A Series of Ten Cases of Malarial Keratitis, with a Report of the Blood Examination," by Dr. Ellett, of Tennessee, published in the *Chicago Ophthalmic Record* for March, 1899, gives a minute description of corneal lesions occurring in malarials similar to those characteristic of the "dendritic keratitis" of Kipp and Van Millingen; but the main interest of the paper lies in the fact that the plasmodium was found in the blood in each case—scrapings from the surface of the corneæ were also examined, but with negative results. In all his cases the disease was monocular. Duration varied from ten days to weeks; relapses, due to fresh attacks of fever, were not uncommon, some patients asserting that their attacks of ague always "settled in their eyes." The paper is an extremely instructive one, and merits careful perusal by all interested in the subject.

(*b*) *Keratitis Profunda.*—Fuchs, in his "Textbook of Ophthalmology," states that "intermittent fever in its chronic form of malarial cachexia sometimes results in a keratitis profunda which is characterised by the absence of marked symptoms of irritation and also by an unusually chronic course." A greyish infiltration of the middle and deep layers of the cornea at or near its centre comes on very slowly, remains stationary for days or weeks, and then subsides without producing any solution of continuity, or in fact affecting the superficial layers at all. Under a lens the apparently homogeneous opacity is found to be made up of minute dots and striæ. Levrier[1] was, I believe, the first to point out the frequent association of this disease with malaria. In one case described by him the patient had suffered severely from malarial fever in Africa. On returning to France the attacks of fever ceased for some time, and then returned in the form of shivering and sweating in the early hours of each morning, while at the same time he experienced a slight sensation of uneasiness in the left eye. Seen by Levrier a month

[1] "Acc. Ocul. dans les fièvres int." Paris, 1879.

later, the upper half of the cornea was found to be opaque, the conjunctiva was injected, and there was slight photophobia ; the patient had shivering followed by a rise of temperature and sweating every morning, exactly the same feverish symptoms as he had suffered from in Africa. Under atropine, fomentations, and quinine in one-half gramme doses twice daily, the eye slowly improved, and eventually the cornea was left almost clear. Similar cases have been described by Sedau, Poncet, Landolt, Javal, and by myself.[1] I have seen in Mr. Morton's clinic at Moorfields a case of diffuse central corneal opacity, looking under the magnifying glass like a shower of snowflakes, in a man who had suffered from blackwater fever on the West Coast of Africa. The patient had had rheumatism, but the symptoms most characteristic of rheumatic keratitis, pain, inflammation, and marked photophobia, were wanting. He did well under atropine and small doses of quinine and arsenic, and the infiltration had begun to subside when he ceased attending and we lost sight of him ; there was no history of syphilis.

(*c*) *Vesicular Keratitis.*—Godo, in reviewing 40 published cases of herpes febrilis of the cornea, says that 13 had occurred in malarials, the corneal eruption being accompanied by herpes of the lips and nose. Tangeman, of Cincinnati, has met with a similar affection amongst malarials, which he calls "keratitis bullosa." I hardly think, however, that this herpes corneæ can be considered as directly due to malaria.

Iritis.

Recorded cases of malarial iritis are not numerous, and in very few is the evidence of malarial origin perfectly satisfactory. While assistant to Mr. Treacher Collins at Moorfields I saw one case of iritis which seemed to us probably due to malaria. The patient was a discharged soldier, married, with three healthy children. He had had "fever and ague" in Burmah and India. When first seen he complained of violent

[1] Soc. Française d'Ophtal., May, 1887. *British Medical Journal*, July, 1900.

pain and photophobia in the right eye. The iris was discoloured, and there was considerable ciliary injection, but no synechiæ, and no fever. He stated that he had had three similar attacks in the same eye in Burmah, that they came on during paroxysms of fever, and subsided in a few days' time under quinine. There was no history of gout, rheumatism, or syphilis. He recovered within a week under atropine, and has not returned to the hospital since. Mr. Collins kindly showed me notes of two other cases he had seen of recurrent iritis in malarials; in both, however, there was a history of gout, and he thought the diagnosis of malarial iritis, though probable, was not absolutely certain. Tangeman's case[1] seems clear enough: his patient suffered from periodic iritis, with pain and photophobia, each attack coinciding with a paroxysm of fever; atropine seemed to have no effect, but quinine gave almost immediate relief. M. Pèchin, at a recent Congress of the Ophthalmological Society of France (1899) read notes of a case of double malarial iritis:—

"The patient, a woman aged forty-eight, suffered from tertian ague in 1870; the fever lasted in all several months, but eventually yielded to quinine. During one of the paroxysms her right eye became injected and painful, with discoloration of the iris and immobile pupil; eventually the iritis subsided, leaving a posterior annular synechia. Five years later she suffered again from a severe attack of malarial fever; this time the left eye became affected in one of the paroxysms, and several synechiæ formed; she has had no recurrences of iritis or malaria since. Dr. Pèchin went carefully into the history and antecedents of the case, and came to the conclusion that the disease was undoubtedly due to paludism."

With reference to this subject, Gower's words ("Medical Ophthalmoscopy," 3rd edit.) are worth bearing in mind:—

"Purulent affections of the eye (chorioiditis, iritis, etc.), such as are seen in pyæmia, have been described in intermittent fever, but are extremely rare, and some

[1] *Cincinnati Lancet*, 1889.

doubt may be felt regarding the diagnosis of the original disease when it is remembered how closely some cases of pyæmia simulate intermittent fever. Even the influence of quinine, on which diagnostic weight is often laid, is not entirely conclusive; for example, in a case of this kind described by Landesberg, although quinine cut short the affection, abscesses formed during convalescence in one toe and forearm."

I hardly see how the formation of abscesses in the case cited invalidates the diagnosis of malaria, as boils and abscesses are very common during convalescence from malarial fever. However, it is always well to remember the possibility of confounding pyæmia with intermittent fever.

Cataract.

The only instances of cataract attributed to malaria which I have been able to find are two cases recorded by Bagot,[1] of Guadeloupe. The first patient was a mulatto boy of fifteen, who had a severe bilious remittent fever with gastro-intestinal symptoms and coma; this lasted two or three days, and immediately afterwards his sight began to be affected; three months later it was found that he had a soft cataract in each eye. The second patient was a mulatto girl of sixteen, who also had an attack of grave malarial fever lasting three days; immediately afterwards sight began to fail; nine months later she also was found to have a soft cataract in each eye.

In the absence of more precise details, it seems hardly fair to comment on the above; but, judging from the published facts alone, I am inclined to think the coincidence of malaria and cataracts must have been accidental.

Anomalies of Accommodation.

My friend Dr. Manson has shown me notes of a case of monocular ciliary spasm associated with malarial aphasia which he saw in Hong Kong; with the cure of the malaria the spasm disappeared. So far as I am aware this is the only case of the kind ever noted; the unilateral character of the spasm is exceedingly curious.

[1] *Brit. Med. Journal*, Epitome, Feb. 20th, 1892.

Cases of malarial cycloplegia have also been recorded by Manhaert, Bull, and others.

Treatment of Malarial Eye Affections.—The treatment is the treatment of malaria. Most essential of all is early removal to a non-malarious climate. Iodide of potassium may be of service in promoting absorption of vitreous opacities.

Quinine Amaurosis.

Wherever malaria is prevalent, there too will cases of quinine amaurosis be frequent, owing to careless administration of the drug.

What is quinine amaurosis? Briefly, it is a temporary blindness, which leaves on its subsidence certain permanent defects of vision, the chief being contraction of the visual fields, diminished light perception, and diminished colour sense; caused by a toxic dose of quinine,[1] producing in succession spasm of the retinal vessels, then breaking down of the less resistant layers of the retina from the diminished blood supply, and, finally, an ascending degeneration of the optic nerve fibres. Granting the word "temporary" a somewhat wide signification, the above may be accepted as an accurate and fairly complete definition.

For the last seventy years visual disturbances due to quinine have been recorded by writers—in fact, almost since the first introduction of the drug. Unfortunately, however, until comparatively recently malarial eye affections and quinine amaurosis have been confounded together, visual defects being attributed to malaria which were too often the result of quinine. At the present day such confusion ought not to exist; the points of distinction between quinine amaurosis and the group of affections commonly included in the term "malarial amaurosis" are many and clearly defined.

At one time it was thought possible that the effects on vision might be due not only to quinine, but also to

[1] The word "toxic," here and throughout, refers to "visual toxicity," if I may be allowed to coin a clumsy but expressive phrase.

diseases for which it was prescribed ; but the uniformity of the signs and symptoms of quinine amaurosis in all kinds of diseases, its repeated occurrence in healthy people who have taken quinine by mistake, and the experiments of Barbaschew on human beings, and Brunner, De Schweinitz, and Holden on dogs, have finally disposed of this theory, if it was ever seriously entertained.

Any of the alkaloids of cinchona appear capable of producing toxic effects, though sulphate of quinine is probably the most active. One case of amaurosis following the administration of tincture of cinchona is recorded.

It is impossible to fix definitely the amount of quinine which will produce amaurosis, as individual idiosyncrasy comes so much into play. It has been produced by doses amounting to only fifteen grains in the twenty-four hours ; on the other hand, as much as an ounce in the same time has had apparently no ill effect—probably through non-absorption. De Schweinitz mentions that he himself has suffered from transient amaurosis after fifteen grains in twenty-four hours, and I have had a precisely similar experience in China. A single large dose seems to be less dangerous than repeated doses reaching a large total in the twenty-four hours, and it is always advisable to remember the liability to relapse after quite small doses.

It may be safely asserted, however, that single doses over twenty grains, or repeated doses amounting in the aggregate to more than a drachm in the twenty-four hours, are always dangerous. Personally, I have never given more than fifteen grains as a single dose, or more than forty grains in twenty-four hours. I have never seen the slightest benefit from larger doses, but, on the contrary, have often seen harm. A high authority on the subject, Dr. Patrick Manson, writes thus :—

"There is a great difference of opinion and practice about the dose of quinine. Some give thirty grains at a dose, some give three. The former, in my opinion, is too large a quantity for ordinary cases, the latter too small. I believe that nothing is gained by excessive doses ; in ordinary circumstances thirty grains spread over two or three days is usually amply sufficient to check an inter-

mittent. If a supposed ague resists the doses of quinine mentioned, *it is advisable to revise the diagnosis.*" (The italics are mine.)

It is difficult to characterise in language of judicial moderation the action of qualified medical men who prescribe quinine in eight or twelve gramme single doses, and even reach the astounding total of *forty-two grammes* in the twenty-four hours; one may, however, be permitted to hint at the likelihood in the near future of such cases possessing a medico-legal as well as an ophthalmological interest.

The general evidences of cinchonism—throbbing headache, ringing in the ears, and deafness—are well known. Shortly after the onset of these symptoms patients often complain of dimness of sight, without perhaps giving the symptom much prominence; and should the eyes be examined in such cases probably nothing will be found save slight diminution of light perception, some loss of accommodation, and sluggish reaction of pupils to light. No serious results need be anticipated from these symptoms; but they should serve as a warning to discontinue the quinine, or at least to greatly reduce the doses.

A well-marked case of quinine amaurosis is, however, very different. Blindness, more or less complete, comes on with startling suddenness. In a case described by me,[1] the patient said "his sight went all at once," and in Browne's case the sudden loss of sight was likened to "turning out the gas." In severe cases there may be absolutely no perception of light; in cases of medium severity the patient can just distinguish day from night.

On examination during this stage the pupils are found widely dilated and irresponsive to light; anæsthesia of the cornea and conjunctiva is occasionally present; in one recorded case there was increased tension. Nystagmus is noted as an occasional concomitant by several writers; I have seen this curious symptom (rotatory) in one case. The media are clear; Geshwind described vitreous opacities in one case of quinine amaurosis, but it is doubtful if they were attributable to quinine. The fundus changes are well

[1] *Journal of Tropical Medicine*, Sept., 1899.

marked and characteristic, the disc is of a dead whiteness, the retinal vessels reduced to threads, the fundus generally pale. Occasionally the macula stands out vividly as a red spot, and the appearances simulate those of embolism of the central artery.

The stage of complete amaurosis varies in duration from a few hours to several weeks; only one case of permanent blindness has been recorded.

The return of vision is slow, and in too many cases very incomplete, contracted fields and impaired light and colour sense remaining permanently. Where amaurosis has been complete some impairment of vision always remains; and even in cases of moderate severity some contraction of the fields and diminution of light sense are apt to persist, though central vision has been completely restored. Where there is permanent visual disability pallor of the discs and contraction of the vessels remain to a greater or less extent.

The contraction of the field tends to assume the shape of an ellipse with the long diameter horizontal (Knapp). In very severe cases—as in my case—the fields are so reduced that only "telescopic" vision remains.

The ophthalmoscopic appearances sometimes closely resemble those of advanced optic atrophy, occasionally those of embolism. Taken in connection with the history and symptoms they are so characteristic that an error in diagnosis should not occur. The points of resemblance between quinine amaurosis and any of the malarial eye affections are few; but, of course, it may occur in eyes already injured by malaria.

Prognosis is, on the whole, good as regards the restoration of central vision, but some—it may be slight—permanent contraction of field and diminution of light perception may nearly always be expected. The liability to relapse on administering quinine, even in small doses, should never be forgotten.

Thanks to the brilliant experimental researches of De Schweinitz and Holden, the pathological anatomy of quinine amaurosis has been worked out with remarkable precision and completeness. Owing to considerations of space, only a brief summary of the results arrived at can be given.

De Schweinitz produced quinine blindness in dogs, noted the exact reproduction of the clinical picture in human beings, and finally submitted, at different stages of the blindness, the optic nerves, chiasms, tracts, and visual centres to microscopic examination. The result of this examination is best given in his own words :—

"In *résumé* I may say in regard to the microscopical appearances that there are thickening and changes in the walls of the optic nerve vessels (endovasculitis); organisation of a clot, the result of thrombosis, an organisation which has been carried on even to the extent of its being channelled by new vessels ; widening of the infundibulum of the vessels as the result of the constriction of the surrounding nerve fibres, causing appearances not unlike a glaucomatous excavation ; and, finally, practically complete atrophy of the visual path, including the optic nerves, optic chiasm, and optic tracts as far as they could be traced.

"It seems, then, very likely that the original effect of quinine is upon the vaso-motor centres, producing constriction of the vessels; that finally changes in the vessels themselves are set up, owing to an endovasculitis; that thrombosis may occur, and that the result of all these is an extensive atrophy of the visual tract."

Quite recently Holden, working on the same lines with new and improved methods, has been able to demonstrate that the changes in the optic nerves and tracts observed by De Schweinitz were secondary to changes in the retinæ. Retinæ examined on the third day after toxic doses of quinine revealed changes in the ganglion cells and in the nerve fibres ; on the ninth day these changes were well marked. Not until the seventeenth day were changes in the optic nerves noticed—breaking down of the medullary sheaths of many fibres. By the forty-second and forty-seventh days the ganglion-cell and nerve-fibre layers of the retinæ were almost gone, and the degeneration of the optic nerves could be traced up to the termination of their fibres in the brain.

The following, therefore, would seem to be the order in which the pathological processes set up by a toxic dose of quinine manifest themselves :—

(1) Constriction of the retinal vessels.

(2) Breaking down of the less resistant layers of the retinæ as a result of the restricted blood supply.

(3) An ascending atrophy of the optic nerve and tracts.

Quinine must be stopped, or at all events given very cautiously in greatly lessened doses, the moment symptoms of amaurosis appear. Given in the doses recommended by Dr. Manson the drug is potent for good, and impotent for evil except in rare cases of idiosyncrasy.

The temporary good effect of inhalations of nitrate of amyl in established cases is remarkable I have had repeated opportunities of verifying it. A moment or two after the inhalation the discs become rosy, the fundus of normal colour, and the patient can perhaps read two lines of type lower than before. Unfortunately these effects are not permanent. Nitro-glycerine given every other day, combined with general tonic treatment, affords the best prospect of ultimate success in restoring, partially at any rate, the visual functions. The patient should, if possible, be removed from the malarious country, in order to avoid the necessity of administering quinine.

CHAPTER X.

TROPICAL EYE DISEASES (NON-MALARIAL).

XEROSIS.

Xerosis of the conjunctiva, with its almost invariable concomitant night blindness, is a common disease in tropical and subtropical countries, and by no means unknown in England. By xerosis is meant a disease of the interpalpebral portion of the conjunctiva, mainly affecting a small area on the temporal and nasal sides of the cornea, by which the conjunctiva becomes dull, greasy, covered with a dirty grey foam, and incapable of being wetted by the tears. Microscopical examination of the xerotic patches shows that the epithelium is greatly thickened, and that each epithelial cell is filled with minute drops of oil; to this fact, and also to the adhesion to the surface of the epithelium of the meibomian secretion, is due the peculiar greasy, frothy appearance so characteristic of the disease. Rod-shaped bacilli, with rounded ends, the so-called bacilli of xerosis, are found in profusion on the surface of the epithelial cells. Mr. Sydney Stephenson, in this country, has never yet failed to find them in cases of epithelial xerosis, not only in cover-glass preparations, but also in cultures made from the frothy patches.[1] As these bacilli, however, are also found in the conjunctiva under certain other conditions, we are hardly as yet justified in doing more than noting their apparently invariable presence in xerosis.

A severe form of xerosis, in which the disease spreads to the cornea (keratomalacia), which frequently suppurates, is unfortunately not uncommon in the tropics, especially in the native quarters of great towns such as Bombay, Calcutta, and Hong Kong, and seems to be the

[1] "Transactions of Ophthalmological Society of the United Kingdom," 1898.

local expression of some obscure general disease; patients, mainly children, emaciate rapidly, suffer from diarrhœa, and often die. Of this class of cases Captain Herbert, of Bombay, exhibited some interesting photographs and microscopic preparations at the Ophthalmological Society in 1899. He attributes xerosis to faulty feeding; it is so common in Bombay amongst the poorer classes that in four months, June to September, 1896, he was able to tabulate one hundred consecutive cases.

A condition somewhat resembling xerosis is often seen associated with ectropion and chronic trachoma, and has been called "secondary" xerosis by Leber, but the resemblance to xerosis is only superficial.

Of the visual phenomena associated with xerosis, the most constant and remarkable is *night blindness*. The expression "night blindness," though convenient, is not a very happy one, inasmuch as the defect of vision develops when illumination is diminished from any cause—in an artificially darkened room, for example. We are all familiar with the difficulty experienced in seeing objects at first on entering a darkened room. In night blindness this defect of vision, transient in the normal eye, is persistent, owing to the retina having lost its power of "adaptation," and no longer acting except to strong stimuli—a condition known as "torpor retinæ." Förster's photometer furnishes a means of estimating the light sense, and hence the degree of night blindness; but the apparatus is clumsy, and seldom used in this country. The amount can be estimated sufficiently well for all practical purposes by testing with faint test types in a darkened room, and comparing the types read by the patient and those read by the surgeon.

It is now generally recognised that night blindness is an almost constant symptom in xerosis, both probably being expressions of malnutrition of the eye. Mr. Sydney Stephenson, in the able and exhaustive paper previously alluded to, quotes many striking examples of this association. Bitot examined twenty-nine cases of night blindness, and in every case found spots of xerosis —the so-called hemeralopic spots.[1] Villemin confirmed

[1] *Gazette Hebdom.*, Paris, May 1st. 1863.

this by investigations amongst soldiers at Strasburg[1] Kuschbert and Neisser described an outbreak of night blindness in a Breslau asylum in which xerosis was present in every case,[2] and many other instances could be given. It must be remembered that the symptom must be looked for, as patients often fail to draw attention to it. I saw many cases of xerosis, both mild and severe, when in charge of the Government Civil Hospital in Hong Kong, but never detected night blindness, as at the time I was not aware of its being an almost invariable symptom. Cases of night blindness without xerosis and without any other discoverable cause (such as retinitis pigmentosa) undoubtedly exist, but are extremely rare ; I have only seen one such case at Moorfields.

It has been suggested that night blindness is due to molecular changes in the retinal pigmented layer, brought about by impoverishment of the blood in the chorioid, and that the xerotic patches are due to degeneration of the superficial layers of the conjunctiva forming a *nidus* for the xerosis bacillus. "In this view both night blindness and xerosis would be attributable to a common cause, viz. impaired nutrition ; while the former would be proximately due to dazzling, and the latter to the bacillus" (Stephenson).

Mr. Stephenson believes that reduction and transposition of the red and green fields of vision is another constant phenomenon in xerosis, having found it in every case which he investigated fully. No fundus changes can be detected.

The etiology of xerosis is still obscure. Its frequent association with malaria and widespread prevalence in malarial countries have led many to believe in its malarial origin. On the other hand, xerosis is found in countries where malaria is unknown—for instance, in schools and asylums in England.

On the whole it appears probable that xerosis is an expression of a state of general malnutrition which may be due to several causes, chief among which are malaria

[1] *Gazette Med.*, May 1st, 1863.
[2] *Brez. Arzt. Zeits.*, 1883, No. 4.

and insufficient or improper food. It is most frequently found amongst the insufficiently nourished classes, and is stated to occur in an epidemic form in Russia during and after the long Easter fast. Dazzling by bright light often seems to act as an exciting cause; hence its frequency amongst sailors, by whom it is often attributed to the malign influence of the moon.

Cases of xerosis always improve *pari passu* with improvement in the general health; with good food, fresh air, and general tonic treatment most cases do well. Keratomalacia must be treated on the same principles as other forms of corneal ulceration. Nesnamoff treats the xerotic patches by washing the conjunctiva and cornea first with normal saline solution, and then with ether. This removes the fat from the diseased spots, and they become capable of being wetted by the tears. He also employs inhalations of ammonia to increase the lachrymal secretion, but the utility of this procedure is questionable. The real cause of xerosis is not deficiency of the lachrymal secretion, and it has never been known to occur after extirpation of the lachrymal gland.

PTERYGIUM.

Most practitioners in tropical countries are familiar with that peculiar affection of the conjunctiva, or, more correctly speaking, of the cornea, which has been somewhat fancifully called pterygium (πτερύγιον = a little wing); nevertheless, some confusion still prevails as to its exact nature, and many confound it with the so-called "false" pterygium or "pterygoid"—a condition to which it bears a superficial resemblance. The etiology, pathology, and treatment of the two affections are quite different, so that it is of considerable practical importance to be able to draw a sharp line of demarcation between them.

A pterygium affects only the inter-palpebral surface of the eye, and is a triangular fold of conjunctiva, the apex of which extends a variable distance into the cornea, while the sides widen out into the bulbar conjunctiva without any very distinct limiting line. In a progressive case this fold of conjunctiva has a reddish

colour due to numerous vessels converging from base to apex. The apex or "head" of the pterygium is well marked, and inserted on and into the cornea like a tendon; to this succeeds a contracted portion called the "neck," the edges of which are rounded and incurved so as to permit the passage of a fine probe a short distance (never entirely) underneath them; the remaining fan-shaped portion is known as the "body." The most usual situation is the nasal side of the cornea. I have never seen a *single* temporal pterygium—when one exists in this situation it is always consecutive to one on the nasal side of the same eye; such double pterygia are by no means uncommon. Both eyes are frequently affected, and I remember one case in which four pterygia co-existed, one on the inner and one on the outer side of either cornea. A true pterygium is due to the slow transformation of a pinguecula (a spot at the inner and outer margins of the cornea where the conjunctiva gradually becomes yellow and thickened from exposure); so-called pterygia at the upper and lower corneal margins belong to the class of "false" pterygia above alluded to.

Not only is pterygium a deformity, but as it progresses it produces more serious inconveniences, such as impaired motility of the eye, epiphora, and defect of vision. The latter may be due to (1) dragging on the cornea, producing irregular astigmatism, (2) impaired motility with resulting diplopia, or (3) invasion of the pupillary area by the head of the pterygium.

The extent of a pterygium depends on its duration. Commencing at the edge of the cornea, it slowly pushes on, dragging the hypertrophied conjunctiva after it, until it reaches the centre (progressive pterygium); finally, it appears to become stationary, and then slowly atrophies to a thin membrane (retrogressive pterygium). A noticeable feature, and one of the many distinctions between it and "pterygoid," is the fact that its progress is unaccompanied by any signs of inflammation.

The disease is comparatively rare in England and in temperate climates generally. On the other hand, it is very common in tropical and sub-tropical countries; I

saw a large number in Hong Kong, chiefly amongst Indian residents, but a few also amongst Chinese.

Treatment.—The head, neck, and a wedge-shaped portion of the body should be excised, and the sooner the better—if possible before the pupillary area is invaded. The little operation I am in the habit of performing resembles that advocated by Fuchs, and answered very well; it consisted in dissecting off the neck and a triangular segment of the body, *pulling* out the head, and then bringing the edges of the conjunctival wound together with a couple of sutures; for this last procedure liberating incisions are often necessary. I have seldom seen recurrences after this operation, and believe the rare cases were due to want of care on my part in closing effectively the gap in the conjunctiva. Panas recommends touching the corneal wound with a thermo-cautery, but I cannot see any advantage in this proceeding; it increases the subsequent nebula, and I do not think lessens the risk of recurrence.

Pterygoid.—As a result of acute blennorrhœa with marginal corneal ulcer, injuries of cornea by burns, caustics, etc., a fold of chemosed conjunctiva may become adherent to the cornea. This "pterygoid," or false pterygium, presents some resemblance to pterygium, from which, however, it can easily be diagnosed by the history, the inflammation, the staining of the ulcer by fluorescin (if the case be a recent one), the arrest of development on the healing of the ulcer, the absence of the characteristic rim of opaque cornea round the "head," and the variable situation. Often, too, a fine probe can be passed completely under the "neck," showing that the apex of the fold alone is adherent (Fuchs).

Where operative interference with a pterygoid is necessary, it usually suffices to "pick" the head out of the cornea; the fold will then retract and become atrophied.

THE FILARIÆ OF THE EYE.

Filaria Loa.—Of all the parasites which have been found in the human eye and its *adnexa*, undoubtedly the most interesting to students of tropical medicine is

the filaria loa, owing to its being peculiar to a tropical country and to the interesting problems in connection with its life history which still await solution.

Filaria loa owes its name to M. Guyot, a French surgeon, who (in 1778) saw several cases amongst the natives of Angola, in West Africa, and utilised the native word "loa" (worm) in its nomenclature. Prior to this cases had been described by Mougin, who called it a "blood worm," and Bajour, who believed it to be a dracunculus. The first record of the worm, however, is a pictorial one, and shows that it was known at least 300 years ago. Dr. Manson has in his possession a woodcut, reproduced from a quaint old book published in 1598,[1] which portrays in realistic fashion a man drawing a guinea worm from his leg, and a woman undergoing a rude operation for the removal of a filaria loa from her eye.

Since Guyot's paper appeared not more than a dozen cases have been recorded. The literature of the subject is unaccountably scanty, when we consider the length of time that has elapsed since the filaria was discovered, and the fact that it is so common on parts of the West Coast of Africa that the natives are able to diagnose its presence in the eye.

The endemic area of loa filariasis appears to be that part of the West Coast of Africa extending from about 5° above the equator to 15° below it. It is exceedingly common in Old Calabar, Gaboon, and Angola, and affects white residents as well as natives. How far inland the disease extends is unknown; in one of the most recent cases recorded,[2] the patient, a white man, became infected at Talagonga, on the Goa, 120 miles from the sea. Miss Kingsley, the well-known lady explorer, states that nearly everyone on the Ogowe river, near Gaboon, suffers from these worms.

Loa filariasis can only be contracted in this endemic area; cases recognised elsewhere have been in natives of this part of the West Coast or whites who had resided

[1] "Vera Descripta Regni Africani." Pigafetta. Frankfort, 1598.

[2] *Bernard Arch. d'Ophthal.*, Oct., 1898.

there for variable periods. That the parasitism is of long duration is shown by the fact that it was not uncommon amongst American negroes before the abolition of slavery; the slaves were brought to America in sailing vessels which took weeks, and even months, to make the voyage. Cases have been recorded of individuals who had been ten years away from the endemic area still harbouring the worms.

"The male worm measures about 25 to 30 m.m. in length by 0 3 m.m. in breadth; the female, 30 to 40 m.m. in length by 0·5 m.m. in breadth. Both sexes are filiform, cylindrical, colourless, like fine fishing gut, tapering slightly at the head, more decidedly at the tail. The body is dotted over with minute chitinous bosses. The anus is subterminal. The tail of the male is markedly incurvated, and provided with five large papillæ on each side of the anus and two rather short, unequal spicules. The mouth is simple, punctiform, and without armature." (Manson, "Manual of Tropical Diseases.") M. Guyon[1] described a filaria loa, 15 c.m. long, extracted by a French naval surgeon from a Gaboon negro, but this was probably a "filaria inermis." Only two cases are on record (by Argyll Robertson and Bernard) of the removal of an intact male filaria loa from the eye.[2]

The parasite is generally found in the bulbar subconjunctival tissue or beneath the skin of the eyelids, but has also been seen and felt in the subcutaneous tissue of other parts of the body, arms, hands, scalp, thorax, etc. It wriggles about with surprising swiftness, wandering from the eyelid to the eyeball and thence disappearing into the orbit within the space of a few minutes. Beyond a very annoying tickling and slight swelling, it seems to produce no ill effects *per se*; the lachrymation and inflammatory symptoms seen are due to the scratching provoked by the violent itching. The natives of the West Coast say that it manifests its presence for two or three days consecutively, and then disappears, to reappear again in a fortnight; after ten to twenty years it disappears altogether. In this country it only appears in hot weather or in very warm

[1] *Annales d'Oc.* 1864, p. 241.
[2] "Transactions of Ophthalmological Society, 1897." *Op. cit.*

rooms. In Dr. Argyll Robertson's case the patient, a lady, invalided home from Old Calabar with dysentery and fever, was confined to her room for a lengthened period, and during this time the worm was almost constantly moving about in the left eye; when she became well enough to go out of doors the filaria ceased its visits, and only reappeared at long intervals when she sat near a fire. In one case, at least, the filaria has been seen passing from one eye to the other by wriggling beneath the skin of the nose.

In all probability one individual harbours several worms, and sometimes one, sometimes another, comes to the surface.

It is not so easy to fix and extract the worm as one might fancy. It moves about so quickly that, as a rule, many attempts have to be made before success is attained, and even then it is generally crushed or broken in withdrawal. In Argyll Robertson's patient it was seized on the sclerotic by fixation forceps, and withdrawn through a conjunctival incision; in Bernard's, after several unsuccessful efforts, it was removed from under the skin of the upper eyelid in a similar manner.

Nothing is known for certain as to the life history of the parasite; its means of ingress and egress and intermediate host (if any) are still matters of pure conjecture. Manson thinks it possible that filaria diurna is the embryonic form of filaria loa, as the parasites co-existed in one of his cases. He has, however, lately examined the blood in another case of loa filariasis without finding any hæmatozoal embryos; an examination of the blood in Argyll Robertson's case gave a similar negative result. In Bernard's case, unfortunately, the blood was not examined. Undoubtedly the embryos of the female filaria loa closely resemble filaria diurna, and analogy suggests that they circulate in the blood and are taken from it by some suctorial insect acting as intermediate host. It is to be hoped that our *confrères* in West Africa may take up the investigation, and bring to light the method of development and propagation of this parasite.

Filaria Oculi Humani.—Filaria oculi humani is rare, or, at all events, only a very few cases have been recorded. It is by no means certain that the filariæ

which have been found in the aqueous, lens, and vitreous belong to the same species, but in the present state of our knowledge it is convenient for descriptive purposes to assume that they do, and to call this species by the old name, *filaria oculi humani*. To those interested in the filariæ of the blood this parasite is of special concern, in view of its possible relationship to filaria Demarquaii. Dr. Manson, in the course of a recent conversation on the tropical entozoa of the eye, told me he thought it quite possible that the *filaria oculi humani* seen by a West Indian observer (and by Barkan, of San Francisco) is the parental form of *filaria Demarquaii*, the blood parasite recently described by him as common in the West Indian Islands, British Guiana, and New Guinea. This is an expression of opinion on which I am not qualified to pronounce, but I think it right to reproduce it here, as Dr. Manson's conjectures of to-day, founded as they are on an intimate acquaintance with medical zoology and prolonged experience of tropical practice, have a happy knack of becoming the proved facts of to-morrow.

Filaria oculi humani has been seen in the aqueous, lens, vitreous, and beneath the retina. I have never seen a case myself, and can only give brief abstracts of the few recorded cases.

(*a*) *In the Aqueous Humour.*—Only two cases of filaria in the aqueous humour of man are known, though in certain animals, notably the ox and horse, it is by no means uncommon (f. Abildgaard—f. papillosa Rudolphi). The first case was described by Dr. A. Barkan, of San Francisco.[1] The patient was a native of Adelaide, Australia, whence he had recently come to San Francisco. He had suffered from severe inflammation of the eyes —probably granular ophthalmia—ten years previously, when living amongst the Australian aborigines. In the anterior chamber of the left eye a whitish, thread-like, immobile foreign body was seen adherent to the lower part of the iris. Barkan performed a downward iridectomy, and removed the foreign body together with the portion of iris to which it was adherent. Professor Knapp subsequently made a microscopical

[1] *Arch. of Ophthal. and Ot.*, vol. v., 1876.

examination of the worm, and pronounced it a filaria medinensis (?).

The second case occurred in Havana. Dr. E. Lopez[1] saw a parasite in the anterior chamber of the eye of a seamstress, a white woman, aged sixty-one, which had been there, judging from the narrative, from August, 1890, to the following February, without producing any more serious lesion than slight cloudiness of the cornea and mild iritis. The parasite, apparently a nematode, is described as having been white, 0·025 m. in length, slender as a fine thread, slightly thicker at one end than at the other, and very active. At a subsequent examination it was found to have slipped into the posterior chamber, and only part of it could be seen through the pupil. Instillation of atropine proved fatal to the parasite, and as the patient refused operation nothing more could be ascertained about its structure and zoological characters.

(*b*) *In the Lens.*—Filariæ of the lens have been not infrequently found in the frog and the perch (Von Nordmann), but are very rare in man. Professor Blanchard, in his learned and exhaustive work on Medical Zoology, gives details of the few recorded cases.

(*c*) *In the Vitreous.*—In 1858 Quadri[2] was consulted by a woman who complained that she saw a thread constantly moving in front of the right eye. With the ophthalmoscope he saw a living entozoon, 22 m.m. long, 0·22 m.m. wide, thicker at one extremity than the other, twisting about freely in the vitreous. Delle Chiaji, the helminthologist, confirmed the diagnosis.

Fano in 1868 described a moving body in the vitreous of a child of twelve, but it seems probable now that the case was one of persistent hyaloid artery ; the same comment applies to the cases published by Mauthner and Ewersbusch.

J. Santos Fernandez[3] has twice met with nematodes in the human vitreous in Cuba. In each case a little animal could be seen to undulate through a central haze ;

[1] *Rev. de Ciencias Med. de Habana*, Dec. 5th, 1891.
[2] Congrès Ophtal. de Brox., 1858.
[3] *Cron. med.-quir. de Habana*, 1889 and 1882.

in one case the parasite disappeared, and vision became normal.

Filariæ have been found in the vitreous of the dog, hawk, and frog.

(*d*) *Sub-retinal.*—Kahut's case of sub-retinal filaria is, I believe, unique.[1] A man, aged thirty-one, nationality not mentioned, presented himself complaining of a defect of vision in the right eye of two months' duration. On examining the fundus a whitish swelling like a pin's head could be seen in the macular region ; the rest of the eye was normal. Two months later the swelling had increased to the size of the optic disc, and had a greyish spot in the centre, from which a mobile thread-like body protruded into the vitreous. Still later—nearly five months after patient was first seen—pain and photopsies were complained of, and on examination movements in the tumour could be detected. Kahut concluded he had to deal with a sub-retinal entozoon making its way into the vitreous, and decided to attempt extraction. He divided the external rectus, rotated the eye forcibly inwards, and incised the eyeball at a point corresponding to the site of the tumour ; a gush of vitreous followed, carrying with it a little grey lump, which on hasty examination with a lens proved to be a rolled-up worm. The wound was sutured, the rectus reunited, and the patient did well, recovering a certain amount of peripheral vision. The drawing of the parasite in Kahut's paper shows a nematode worm, pointed at one end, blunt at the other, measuring 10 m.m. by 0·3 m.m. Rudolph Lenkart, who examined it, describes it as "a larval filaria or strongylys, or possibly a scolex embryo."

Filaria Inermis.—This filaria has been found in the horse and ass as well as in man. Only three cases in the human eye are known, although it is possible that the worm described by Guyon as a filaria loa (*vide supra*) belonged to this species. One 115 m.m. long, removed from the conjunctiva, is to be seen in the Museum of the Ospedale Maggiore of Milan ; another was extracted from under the bulbar conjunctiva of a

[1] *Knapp-Schweiggers Arch.*, 1892.

woman of sixty by Valada (Blanchard). Angelo Pace, of Palermo, removed one measuring 100 m.m. in length by 1·5 m.m. in breadth from the eyelid. In the horse and ass it is frequently confounded with filaria papillosa.

Of the other entozoa of the eye, such as monostoma and distoma lentis, cysticercus, echinococcus, trichina, I do not propose to speak here, as they cannot be regarded as more prevalent in tropical than in temperate climates.

THE OCULAR MANIFESTATIONS OF LEPROSY.[1]

Eye lesions are very frequent in leprosy, especially in the tubercular form. It has been estimated that the eyes are attacked in 80 per cent. of all lepers. The presence of Hansen's bacillus in all leprous infiltrations of the eye has been demonstrated over and over again, but the mode of its entrance there is still disputed—some contending that infection is from without, others that it proceeds from within. On the whole the balance of evidence is in favour of the latter view; for leprous eye lesions are invariably secondary to cutaneous or nervous lesions, and a careful study of the infiltrates in the eye shows that when the conjunctiva is attacked it is always *after* a subjacent lesion.

In the *lids* we find maculæ and tubercles, which either disappear after a variable period without leaving traces or terminate in atrophic lesions, fall of hair, etc.; the sites of election are the eyebrows and the free border of the lid.

Localisation in the *conjunctiva* is rare, and always secondary to subjacent scleral lesions—a fact conclusively demonstrated by Hansen and Bull. The so-called conjunctival tubercles are nearly always tubercles of the episclera elevating the conjunctiva. The occasional presence of Hansen's bacillus in the conjunctival secretion has long been demonstrated.

The anterior portion of the *sclerotic*, extending from the insertion of the recti to the cornea, with the corresponding episclera, appears to be the seat of predilection

[1] Those interested in this subject are recommended to read the able monograph on "The Ocular Manifestations of Leprosy" by MM. E. Jeanselme and V. Morax, of which I published an English translation in the *Journal of Tropical Medicine*, 1899.

for the primary ocular localisation of leprosy. In every case where the eye is involved this zone is affected either by circumscribed infiltrations, tubercles, and little tumours, or by diffuse infiltrations showing clinically as stainings, vascularisations, or modifications of colour. Subjective symptoms are slight; if pain be present, it indicates iritis or cyclitis.

Anæsthesia of the *cornea* is almost invariably present in anæsthetic leprosy. True corneal disease is also frequent in both forms, and takes the form of infiltration; in one class of cases (the commonest) the process closely resembles interstitial keratitis; in another the infiltrate presents the characteristics of a tumour.

The *iris* is very frequently attacked; the lesions vary a good deal, but the most characteristic form is that in which the whole surface is speckled with tiny grey points. Posterior synechiæ are the rule. Occasionally lepromas of considerable size are found. Signs of cyclitis are rare.

Infection of the periphery of the *retina* and *chorioid* corresponding to the anterior segment of the sclerotic nearly always occurs, but is difficult to demonstrate during life owing to corneal opacities.

CHAPTER XI.

TRACHOMA ("MILITARY OPHTHALMIA").

The ravages of trachoma in standing armies form an interesting chapter in military history. The disease—previously endemic in many countries, notably Egypt—was first brought prominently into notice in Europe in the last years of the eighteenth century ; the soldiers of the army landed by Napoleon in Egypt contracted it there almost to a man, and on their return to Europe spread this "Egyptian" or "military" ophthalmia far and wide amongst other armies and the civil populations.

"In some countries it became frightfully prevalent. In the English Army during the year 1818 there were more than 5,000 on the invalid list who had been rendered blind as a consequence of trachoma. In the Prussian army, from 1813 to 1817, 20,000 to 30,000 men were attacked by it ; in the Russian army, from 1816 to 1839, 76,811 men were subjects of the disease. In Belgium in 1840 one out of every five soldiers was affected with trachoma. The French army, which was supposed to form the starting point of the disease, was just the one that, relatively speaking, was least attacked. The armies disseminated trachoma among the civil population through the discharge of soldiers affected with eye disease, through the quartering of troops, etc. When they had so many trachomatous soldiers in the Belgian army that they did not know what to do, the Government applied to Jüngken, who was at that time a celebrated ophthalmologist in Berlin. He recommended them to dismiss the trachomatous soldiers to their homes. By means of this fatal measure trachoma soon became diffused in Belgium to an extent which has been observed in no other European State." (Fuchs.)

Compared with these appalling figures, trachoma is

comparatively rare in its epidemic form in modern armies; though the "average constantly sick" in our own Army in certain foreign stations is still considerable, and epidemics occasionally break out in Army schools. Complete blindness is now a very rare sequela in military life. This decrease in the disease is due partly no doubt to attenuation of the virus and to more enlightened methods of treatment, but mainly to improvements in housing, ventilation, and sanitation generally, by which infection is reduced to a minimum.

Among civil populations, especially in schools and asylums where human beings are brought together in circumstances favouring close contact, trachoma is still a widespread and terrible disease, and in many countries has called for State interference with a view to its prevention and treatment. After optic atrophy and purulent ophthalmia, it is the most frequent cause of blindness in the total blind population of the world.

That trachoma is infectious there is no possible doubt, but the laws governing its spread by contagion and infection are still obscure, notwithstanding the immense amount of work done on the subject. It is a remarkable fact that it may remain in one eye for years without affecting the other, and that it may affect one member of a family living together without attacking the others; while, again, it often spreads like wildfire through schools, asylums, or other communities.

Of the many problems connected with trachoma—a disease which, as a scourge of the human race, may fitly rank with leprosy, tubercle, and syphilis—perhaps the most interesting is the influence of *race* on its genesis and spread. The literature of the subject is scanty, and many points require further elucidation, but already one great fact stands out sufficiently clearly—namely, that the dominating influence in the etiology of trachoma is race. The labours of Swan Burnett, Chibret, and others show that the races composing the population of the world vary to a most extraordinary extent as regards receptivity of the trachoma virus, and that this variation cannot be explained by differences of civilisation and sanitary surroundings; or even, except in a limited sense, by differences of climate. Hence any attempt to study the

distribution of the disease from a purely geographical standpoint—to construct a "trachoma map" of the world so to speak—is foredoomed to failure; the trachoma problem is one of race, not place. No one can deny the influence of want, overcrowding, filth, and insanitary surroundings on the spread of what is, to a certain extent, at all events, both an infectious and a contagious disease but these influences are entirely subordinate to two great factors—first, race; and secondly, but *longo intervallo,* climate.

RACE.

It can be shown that one race, at least, seems to be incapable of contracting trachoma; that other races enjoy a relative immunity; that others, again, are receptive to an extraordinary degree; and that between the two latter classes come races which are neither immune nor exceptionally receptive.

(1) **Absolute Immunity.**

It appears to be certain that the Canadian indigenous tribes, including the Esquimaux, are entirely immune; both the specialists and the general practitioners of the Dominion are unanimous in their belief in this *absolute* immunity.

This immunity is the more extraordinary inasmuch as these savages live under all the conditions usually considered most favourable to the development and spread of trachoma. They herd together in tiny hovels, their habits are filthy, and their food insufficient; tubercle and syphilis are rife, and epidemic diseases, such as scarlet fever and small-pox, ravage them to an extent unknown amongst civilised peoples.

The Indians of the United States, who do not belong to the same race as the Canadian Indians, are by no means free from trachoma. Indians from the Canadian tribes are found in Pennsylvania, and are presumably immune, but unfortunately we have no information.

(2) **Relative Immunity.**

Pure-blooded negroes, such as those from Senegambia, Guinea, and adjacent parts of the West Coast of Africa,

enjoy a relative immunity as compared with whites; in the United States this immunity appears to be almost absolute. Swan M. Burnett, of Washington, first drew attention to this curious fact as far back as 1876, in a paper read at the International Congress of Ophthalmology in New York. The most remarkable instance of immunity adduced in this communication was that of the negroes employed in railway construction in Tennessee, who, living side by side with white workmen (chiefly Irish), under the same sanitary conditions, remained quite free from trachoma, although the whites suffered very severely.

Since 1876 Burnett has seen two or three cases of trachoma in mulattoes, none in negroes. In his latest report he produces evidence on the subject from the ophthalmic surgeons practising in parts of the States where negro populations are large. Callan, of New York, has only seen one case in a negro. Savage, of Nashville, in a population one-third of which is composed of negroes, has never seen a case amongst them, though it is common enough amongst the whites. Ray, of Louisville, sees 800 to 1,200 cases of eye disease annually amongst negroes, but never trachoma, although many of his white patients are trachomatous. Phillips, of Savannah, White, of Richmond, Ayres, of Cincinnati, have each seen one case. Bruns, of New Orleans, believes he has seen eight, but is doubtful as to some of them. Burnett himself has seen six doubtful cases in twenty years, but has never yet seen entropion.

The evidence from the West Coast of Africa is very striking. In response to my appeal for information, Dr. Renner, who has practised for seventeen years in Sierra Leone, favoured me with the following facts: I quote his words:—

"As surgeon in charge of the Colonial Gaol at Freetown, which has an average of 230 prisoners, who come from the labouring classes of nearly all sections of the heterogeneous tribes forming the population of this colony and its protectorate, I can affirm that there is not a single trace of this disease amongst the inmates of the prison. In children, again, the same remarkable immunity is apparent. In the two large negro schools, attended by

200 boys and 125 girls respectively, not a single case of trachoma has ever been observed—a marked contrast to our own Board schools. Amongst the West Indian negroes from the different West Indian Islands, and their children, in this colony the same immunity is observed."

Dr. Renner concludes by expressing his firm conviction that "the negroes of the West Coast enjoy an absolute immunity from trachoma."

Major Hosie and Captain Smith, brother officers of mine who have served for long periods in West Africa —the former in Freetown and the hinterland up to the Niger source, the latter in Sherbro and the South Mendi hinterland—have both informed me that they have never seen trachoma in West African negroes, though conjunctivitis in various forms is common.

Van Millingen, of Constantinople, disputes the racial immunity of the negro on the grounds: First, that he has seen cases of trachoma amongst negroes in Constantinople; and, secondly, that the indisputable immunity seen in other countries is due not to race, but to improved sanitation. Chibret, however, in his admirable paper on the geographical distribution of trachoma, shows that from the standpoint of ethnology the negroes of Constantinople are derived from a highly impure race, that of North-eastern Africa, which for a lengthened period has intermixed with a (highly receptive) Semitic element, so that the occurrence of cases amongst the people of this mixed race is not only possible but probable. Van Millingen's second objection does not fit in with facts; doubtless, since the abolition of slavery a small minority of the American negroes have made sanitary progress, but the great majority have still much to learn from their white neighbours in this respect.

The Cingalese are stated to be relatively immune (Hirschberg).

(3) Receptivity.

The evidence of the receptivity of the white races is too well known to need recapitulation here. Among whites the Jews, Poles, Italians, and Irish suffer most; and here undoubtedly poverty, defective sanitation, and

overcrowding are the main causes of the increased prevalence of trachoma amongst these races *as compared with other white races.* Egypt, Persia, and India have been ravaged by trachoma for centuries; here, also, defective sanitation plays a certain part, but meteorological conditions are probably the main factors in reinforcing racial predisposition. (See below.)

Some of Van Millingen's statistics are worth quoting, as showing the relative proportion of trachoma to other eye diseases in certain countries.

Percentage of Trachoma to Other Eye Diseases.

England	0·07	Turkey	18·00
Scotland	0·70	Bulgaria	20·00
Ireland	3·00	Russia	25·00
America	3·00	Greece	25·00
France	4·00	Italy	25·00
Belgium	4·00	Portugal	25·00
Holland	7·05	Central Asia ...	45·00
Spain	11·09	Africa	50·00
Hungary	12·00	Roumania	52·00

To the Chinese and Japanese belongs the unenviable distinction of being the most susceptible. Trachoma accounted for no less than 78 per cent. of ocular affections in Southern China; Mujashita, of Tokio, places the percentage in Japan as high as 75. American observers note the same prevalence in the Chinese quarters of their cities. In Java and the Malay Peninsula the Chinese suffer to a much greater extent than the Malays. In the course of a recent conversation with me on the subject, Dr. Manson expressed the belief that the peculiar shape of the Mongolian eye had much to do with the extreme susceptibility of the race to conjunctival troubles; my own experience of four years in China inclines me to the same belief.

CLIMATE AND SUBSIDIARY INFLUENCES.

Meteorological conditions, while they appear incapable of affecting immunity or partial immunity, have an important influence in reducing or increasing the spread of trachoma amongst the susceptible races.

It has long been recognised that, as a rule, the climate of high altitudes is unfavourable to the development and spread of the disease : for instance, trachoma is almost unknown amongst the races composing the permanent population of Switzerland, and cases brought into the country by travellers ameliorate rapidly. The rule, however, has numerous and important exceptions. Treacher Collins, in travelling through Persia, was particularly struck with the enormous amount of trachoma amongst the inhabitants of the mountain chains between Shiraz and Ispahan. River, of Colorado (quoted by Swan Burnett), has seen cases in Leadville at an altitude of 10,000 feet. Viger found it very prevalent at high altitudes in Algeria. Many other instances could be given. These facts have led Van Millingen and Swan Burnett to believe that altitude has little or no influence—almost the only point on which these observers agree. I believe, however, with Chibret, that altitude has an important influence on the disease, but not altitude *per se*. Where extreme heat, dryness, and dust co-exist with high altitudes, as in Persia, Algeria, and Colorado, these conditions overpower the immunising influence of diminished atmospheric pressure ; but, on the other hand, when the air is cold, moist, and dust free, as in the Alps, trachoma becomes rare or unknown.

Strong sunlight, by producing inflammatory conditions of the conjunctiva, is undoubtedly a predisposing cause ; add to this dryness, dust, and national headgear affording insufficient protection to the eyes, and we have all the essentials for the development of trachoma in a receptive race.

Filth, overcrowding, starvation, and all the conditions aptly summed up by Chibret in one word—misery—are also factors in increasing racial receptivity ; they probably act mainly by lowering the powers antagonising disease, but partly, no doubt, by favouring contagion.

DEFINITION AND SYMPTOMS.

Trachoma is a specific infectious disease affecting the conjunctiva, and characterised by inflammation, hypertrophy, and *subsequent cicatricial contraction* of that membrane. The special micro-organism of the disease

has yet to be discovered. The disease originates in infection from another trachomatous eye, through towels, handkerchiefs, dressings, dust, flies, etc.

The patient at first feels only a little irritation, with a feeling as of dust in the eye, symptoms generally attributed to "a cold": the irritation gradually gets worse, and is soon accompanied by photophobia, lachrymation, and a sticky discharge which causes adhesion of the lids when the eye has been closed for any length of time. At this stage he generally consults the surgeon.

On examining the eye or eyes the palpebral opening is found smaller than usual, owing to drooping of the upper lid, and on everting the already thickened and stiffened lids the conjunctiva of the tarsus and fornix is found reddened, swollen, and velvety from the development of tiny papillæ; the bulbar conjunctiva is slightly, if at all, affected. So far there is nothing characteristic about the disease, as these are the symptoms of simple catarrhal conjunctivitis. If, however, the disease is not aborted in this stage by appropriate treatment, the pathognomonic *granulations* (hence the name "granular ophthalmia") soon make their appearance. The granulations grow in the superficial layers of the conjunctival tissue, and resemble in appearance minute sago grains; they are best seen in the fornix, especially in the upper. Histologically each is composed of gelatinous material within a capsule of connective tissue, the latter not well demarcated from the surrounding tissue, and showing a strong tendency to extend. As the case proceeds the granules increase in number, then become stationary, and finally disappear so far as the gelatinous material is concerned, having gradually converted the conjunctiva into cicatricial tissue which, contracting, produces *entropion*, with much deformity and discomfort from the ingrowing cilia. But this is not all; entropion is not the only, nor indeed the most serious, complication. In the course of the disease the conjunctival affection is exceedingly apt to extend from above down over the globe to the superficial epithelial layer of the cornea (which is really a continuation of the conjunctival), producing what is known as "pannus."

Pannus is a superficial vascularity and infiltration of

the cornea, always beginning above, generally confined to the upper half, but sometimes extending over the whole cornea; it seems to bear no relation to the apparent intensity of the disease process in the tarsus, being sometimes marked in otherwise mild cases, slight or absent in severe ones. When intense and extensive the cornea presents the appearance of a pink rag. If not soon absorbed, it undergoes connective tissue alteration, penetrates the cornea proper through Bowman's membrane, leaving it eventually covered with a permanent grey veil and the eye almost blind; or, worse still, the cornea may soften owing to the infiltration, and bulge forward—*ectasia corneæ.*

Ulcers of the cornea form another frequent complication of trachoma; they generally appear as grey or yellowish grey spots at the edge of a pannus, but may develop in clear cornea. A drop of fluorescin instilled into the eye turns their surface a pretty green colour, showing loss of tissue and distinguishing from infiltrations or nebulæ. These ulcers leave permanent opacities (known as "nebulæ" or "leucomata," according to the density of the cicatrix), or may (rarely) penetrate to the anterior chamber, causing escape of aqueous and prolapse of iris.

DIAGNOSIS.

In the very early, or papillary, stage, trachoma is indistinguishable from acute **catarrhal conjunctivitis,** and in those fortunate cases where treatment has been so early and energetic as to cut short the disease in this stage the diagnosis may remain doubtful. Such cases are more frequent than generally supposed. In too many cases, however, the persistence of the discase and development of the characteristic granulations soon remove all doubt as to its trachomatous nature. **Follicular conjunctivitis,** a disease common in children before puberty, is frequently confounded with it, as the so-called "follicles" bear some resemblance to trachoma granules. Commencing as a catarrhal conjunctivitis, this affection soon leads to the development of little yellowish bodies arranged like strings of beads along the lower retrotarsal fold; the progress of the disease is slow; it usually

lasts some weeks, and may last months. The signs distinguishing follicular conjunctivitis include the following: The "follicles" are yellowish, not translucent grey; they are practically confined to the *lower* retro-tarsal fold; the conjunctival tissue is but little affected; there is no ptosis; neither pannus nor ulcers develop (though the ordinary scrofulous, or apparently idiopathic, ulcer may occur coincidently); and there are no cicatricial changes. The treatment of catarrhal and follicular conjunctivitis includes astringent drops (such as sulphate of zinc one or two grains to the ounce) twice or thrice daily, bathing with boric lotion, and the use of dark protection glasses to relieve photophobia. Suspicious cases should be treated as trachoma.

Phlyctenular conjunctivitis is occasionally mistaken for trachoma, though it is difficult to see why, as the resemblance is practically *nil*. It is extremely common in scrofulous children. Its pathognomic lesion, the "phlycten," is a tiny reddish cone, which appears at some part of the *limbus* of the conjunctiva—*i.e.* where the conjunctiva ends at the edge of the cornea, or, to put it more accurately, where the conjunctivæ, scleræ and corneæ join. This cone is composed of lymphoid tissue elevating the epithelium of the conjunctiva; after a time the apex of the cone breaks down, leaving a small ulcer which soon heals. The term "phlyctenular" conjunctivitis is a bad one, as there is nothing vesicular about the so-called phlyctens. The number of phlyctens varies; there are sometimes only two or three, more often several. Their site also varies; they may occur not only on or exterior to the limbus, but internal to it on the cornea, producing on the latter shallow ulcers which heal quickly, often without resulting opacity. Occasionally such corneal ulcers creep, by a process of alternate erosion and healing, right across the cornea in riband fashion, producing what is known as the "vascular fasciculus."

The life of an individual phlycten is a short one, seldom exceeding two or three days. The disease is seldom of a serious nature. Treatment, as a rule speedily efficacious, consists in the measures adopted for ordinary conjunctivitis, supplemented by general tonics,

diet, etc. When the phlyctens show a tendency to become chronic or indolent, mildly irritant applications, such as insufflated calomel or the dilute yellow ointment, are often of great service.

Blepharitis, or marginal inflammation of the lids, is a frequent accompaniment of trachoma, but is often seen alone. Small pustules or scabs form at the roots of the lashes; the latter soon fall out if the disease be neglected. The scabs or scales should be removed by bathing with a hot soda solution, after which the dilute yellow ointment is applied. This must be done daily, or twice daily, until a cure is effected.

A condition somewhat resembling *xerosis*, known as "xerophthalmus" frequently develops in the later stages of long-standing cases of trachoma in which the conjunctiva is almost completely atrophied. Similar greasy, dry-looking spots appear in the eye, and the retrotarsal folds of conjunctiva almost wholly disappear (symblepharon posticus). When pannus has complicated such cases, the eyes become not only incurably blind, but a source of intense discomfort owing to the feeling of heat and dryness, the entropion and ingrowing cilia.

PROGNOSIS.

The prognosis of untreated or neglected trachoma is, as may be gathered from the description of the disease and its complications, about as bad as can well be imagined. When, however, early and energetic treatment is instituted, and *continuously and efficiently carried out*, the prospects of complete recovery in the vast majority of cases are bright; though it must be confessed that cases do occur—seldom, however, in the well-to-do classes—in which the most careful treatment seems to have little or no effect, and the unfortunate patients steadily go from bad to worse, burdens to themselves and their friends.

TREATMENT.

The measures which should be taken in all well-conducted schools to ensure the early detection and treatment of children with defective vision, and the isolation of those suffering from incipient trachoma,

constitute one of the most important branches of *school hygiene*, and in this direction most important developments have taken place in recent years. Many, if not most, of our large charity schools, orphanages, etc., are now regularly inspected by ophthalmic surgeons; and in some of the largest a most excellent system is in vogue by which trachomatous children are segregated in isolated buildings apart from the rest. Army medical officers should make a point of invariably inspecting the children's schools in barracks during working hours at their weekly inspections, and taking such steps as are possible in the way of isolating children with sore eyes. It is a useful thing also to issue test types, with a few simple instructions, to teachers, with directions to send for inspection any children presenting symptoms of inflamed eyes or defective vision. The men's eyes should also be inspected at the weekly "hands and feet" inspection, and cases of trachoma amongst them should be treated in separate trachoma wards in hospitals.

In the **early or pre-granular** stage the sheet-anchor of treatment is free brushing of the everted lids with ten grains to the ounce nitrate of silver solution once in the twenty-four hours; this should be combined with frequent and free irrigation with warm 1 in 5,000 corrosive sublimate solution *by a trained attendant*. It is difficult to over estimate the value of this means of treatment in the "papillary" stage of trachoma; when thoroughly and efficiently carried out it is often successful in aborting the disease. The application causes a good deal of pain, which unfortunately is but little relieved by subsequent instillation of the ordinary cocaine solution, owing to the formation of an insoluble chloride of silver preventing the anæsthetic action of the cocaine. Dr. Neuschuler, of Galezowski's clinic, has, however recently drawn attention to the valuable properties of *azotate* of cocaine for this purpose; this salt forms no precipitate with nitrate of silver and a few drops of a 1 in 20 solution instilled in the conjunctival sac before the application of the caustic renders the little operation painless. When the inflammation and hypersecretion of this stage have disappeared under the silver treatment, it may be

advantageously replaced by daily *light* touching of the hypertrophied conjunctiva with solid sulphate of copper, supplemented by two grains to the ounce sulphate of copper drops twice or thrice daily until the cure is complete.

In the very early **granular** stage also the silver treatment should be tried, and will generally be found efficacious. Pannus or corneal ulcers do not contra-indicate its use, but in the case of ulcers great care should be taken to prevent the silver coming into contact with the cornea, and in neutralising the excess of silver by means of salt solution.

When, however, the granulations have been present for any length of time, nitrate of silver often fails in its effect, and more energetic measures directed mainly to the destruction of the granulations must be instituted. Of the various measures advocated with this view the one I am most familiar with, and have seen followed by the happiest results when thoroughly and conscientiously applied, is *expression by roller forceps*, as advocated by Dr. Knapp, of New York. The instrument used is known as "Knapp's roller forceps," and is a small pair of forceps with grooved rolling cylinders at the ends instead of points. The technique of the operation is best given in Dr. Knapp's own words :—

"To express trachomatous infiltration both thoroughly and with the proper care not to wound the cornea nor bruise or lacerate the conjunctiva is painful and requires time. I therefore, as a rule, use general anæsthesia. As the operation has proved harmless, I operate on the four lids at the same sitting. The upper lid when everted should be seized at its edge with the roller forceps at the centre of the retrotarsal fold, drawn upwards, so that the whole extent of the granular deposition shall be exposed. Then the lid is held everted with the index finger, and one cylinder is pushed deep into the upper fornix over the edge of the lower lid, which, remaining *in situ*, protects the cornea. The other cylinder passes over the tarsal surface of the conjunctiva. The forceps is now compressed with moderate force and drawn forward, so that we notice the even rolling of the cylinder that lies on the tarsal

surface. The granules come out more or less crushed by the cylinders, and if they are soft their contents are seen only as a gelatinous liquid. The forceps is reintroduced, and the manœuvre repeated over and over again until all trachomatous substance is pressed out. At first the tissue caught by the rollers is thick and resistant, but gradually it thins down, and when all the infiltrated substance is out the retro-tarsal fold stretches as a thin doubled-up membrane between the cylinders. Then the centre of the fold should be drawn up and inward, so as to unfold and stretch out the portion concealed behind the outer commissure. This portion is then pressed out with the roller as thoroughly as the central portion. The same ought to be done with the lower lid and the caruncle. If, which is not rare, the tarsal surfaces of the lids are also beset with granules, these can best be pressed out if, on the lower lid, one cylinder passes over the outer and the other over the inner surface. On the upper lid this need not be done, as the longer and thicker tarsus gives sufficient resistance for the roller to liberate also the tarsal surface from the included granules. The impressions from the ridges of the cylinder give to the surface a fluted appearance which must be uniform, that is, free from granules. On inspection the whole retro-tarsal fold, with the canthal portions, should be entirely free from granules, and present a dark red surface with a number of small redder dots, apparently the cavities of the granules now filled with blood. The surface may or may not be washed over with a small pad of absorbent cotton dipped in a 1 in 500 solution of bichloride of mercury."

The inflammatory reaction is slight; nothing further need be done beyond bathing the eyes with antiseptic solution, and perhaps touching lightly with blue-stone once or twice a week for a few weeks.

Curetting is another procedure much lauded by its advocates. Each granulation is evacuated and its capsule scraped out with a sharp spoon. I have never seen curetting performed, but I fail to see what advantages it can possess over expression.

Brossage (*anglicé* "brushing") consists in brushing away the granules with a very stiff brush, and the

mopping over the raw surface with 1 in 500 corrosive sublimate. In a case of trachoma with an extraordinary abundance of granulations which I had under my care in South Africa (the only bad case, by the way, which I met with in the Army during the campaign) I tried a modification of this method—scarifying freely with an ordinary needle, and then rubbing with 1 in 500 corrosive sublimate solution—with complete success, though the pain and inflammation were at first rather alarming; my only reason for adopting the treatment then was that I had no roller forceps in my possession.

Excision of the granular conjunctiva has been, and is, performed in severe cases, chiefly in Paris (Galezowski) and Russia. Mr. Bitzos, of Cairo, advocates, and has actually carried out, dissecting out the tarsus and replacing it *inverted*, in similar cases; in cases of marked severity and chronicity, in patients indifferent to cosmetic considerations, he removes the tarsus altogether, on the assumption that the tarsus "possesses an anatomical aptitude for the production of trachoma."

Pannus and ulcers require no special treatment apart from the general treatment of the disease.

The fact that cases of old-standing trachoma with bad pannus have sometimes showed great amelioration after an intercurrent attack of purulent ophthalmia, led to the "jequirity" treatment, still warmly advocated by De Weeker,[1] who asserts that "thousands of blind have recovered their sight" by this means. The infusion of jequirity seeds produces violent conjunctival inflammation, on the subsidence of which the pannus will often be found to have practically disappeared; *per contra*, however, many cases have been recorded in which extensive corneal ulceration, and even complete loss of the eye, have followed its use, and the treatment, at all events in England and Germany, seems to have been now practically abandoned.

[1] *Annales d'Oculistique*, August, 1901.

CHAPTER XII.

INJURIES OF THE EYE.

Owing to the very nature of the soldier's profession, involving as it does the carrying about with him, using, cleaning, and keeping in order weapons of destruction, wounds and other injuries of the eye are common in military practice, even in times of peace, though not nearly so common as amongst civil populations engaged in certain trades, such as those connected with the preparation of iron and steel. In war the percentage of eye injuries directly due to war, as compared with war injuries of other parts, has varied considerably in different campaigns, but has never been so large as one would be inclined to expect. The following figures, quoted by Fuchs from Reich, give the numbers and percentages in three great campaigns :—

	No. of Wounded.	Eye Injuries.	Per Cent. of Eye Injuries.
War of the Rebellion ...	408,072	1,190	·29
Franco-German War ...	75,321	464	·61
Armenian War	13,091	290	2·21

The annexed table, taken from that wonderful store-house of information "The Medical and Surgical History of the War of the Rebellion," gives some interesting data concerning the 1,190 cases of gunshot wounds of the eye which occurred in that long and terrible campaign.

Table of 1,190 *Cases of Gunshot Wounds of the Eye.*

Extent of Injury.	Cases.	Died.	Duty.	Dis-charged.	Un-known.
Destroying sight of both eyes	63	17	...	44	2
Destroying sight of right eye	393	12	87	286	8
Destroying sight of left eye	387	24	95	258	10
Destroying sight; side not given ...	45	11	9	17	8
Injuring sight of right eye	25	...	9	13	3
Injuring sight of left eye	20	...	8	8	4
Injuring sight; side not stated ...	6	...	1	2	3
Undetermined cases; right eye ...	106	...	71	24	11
Undetermined cases; left eye ...	116	...	83	20	13
Undetermined cases; side not stated	29	...	16	7	6
Aggregate	1,190	64	379	679	68

For practical purposes the best mode of classifying eye injuries is to divide them into penetrating (*i.e.* penetrating the globe) and non-penetrating.

(*a*) CONTUSIONS (WITHOUT RUPTURE OF GLOBE) AND NON-PENETRATING WOUNDS.

(1) **Lids and Orbit.**—Injuries of the lids and orbit are very common, but are more likely to be treated by the general surgeon than the specialist. *Ecchymosis* of the lids may be due to direct or indirect injury; a familiar example of the former is the ordinary "black eye," which often looks a very terrible injury, and may take two or three weeks to subside. It occasionally ends in abscess of the lid, but as a rule temporary disfigurement is the only inconvenience. Sometimes extravasations of blood take place in the back of the orbit, producing exophthalmus, and they have been known to cause atrophy of the optic nerve by pressure on it. Ecchymosis of the lids from indirect injury, *e.g.* fracture of the base of the skull, is a serious sign; it is not accompanied by swelling, and does not make its appearance for some twelve or more hours after the injury. *Wounds* of the lids and orbit must be treated

on general surgical principles; their cicatrisation is very apt to lead to serious deformity, such as ectropion, etc. *Fracture* of the margin of the orbit may follow a very severe blow; if seen at once, the resulting unevenness renders diagnosis easy; later the fracture may be masked by swelling. A very serious form of fracture of the upper orbital wall may be occasioned by a pointed body, such as a stick, pencil, pipe, etc., being driven through it; its diagnosis and treatment are discussed at length in text-books of general surgery, and need not be entered into here. *Emphysema* in the lids or in the orbital cavity, causing exophthalmus, is an alarming looking symptom; it is greatly increased by blowing the nose or other strong expiratory effort. It invariably signifies fracture into a neighbouring cavity. In many cases it is impossible to discover the exact seat of fracture; Walser's experiments, quoted by Fuchs, show that in these cases the lamina papyracea of the ethmoid bone is the part affected; the entire contents of the orbit are forced back by the blow, and the portion of the bony wall alluded to is so thin that it gives way. Treatment consists in a pressure bandage; the patient should be instructed to avoid strong expiration.

(2) **Displacements of the Eye.**—*Exophthalmus* may be caused by emphysema in the orbit; it is easily diagnosed from that due to extravasation by the presence of other symptoms of emphysema, and by the fact that the eye can be pushed back. Such cases generally do well under a pressure bandage without leaving permanent ill effects; but prognosis just at first should be guarded, as paralysis of one or more ocular muscles, and even blindness through injury of the optic nerve, have been known to follow. Paralysis of ocular muscles from contusion of distant parts of the skull are occasionally seen, the nerve most frequently affected being the abducens. Rupture of the carotid artery into the cavernous sinus, the result of bad basal fracture, produces the remarkable condition known as *pulsating exophthalmus*. The eye is proptosed by a tumour which can be felt and even seen pulsing, and the patient complains of a constant rushing sound in his ears; the conjunctival veins are large and tortuous, and the

veins of the fundus are also enlarged, otherwise the eye itself appears normal, and vision may be quite unaffected ; some cases, however, end in optic neuritis and blindness. Digital pressure of the carotid of the same sides stops the pulsation and markedly diminishes the size of the tumour. The treatment is that of aneurism, viz. systematic digital compression or ligature of the common carotid.

The *tension* of the eye is often noticeably reduced by a blow, without any apparent lesion ; this curious sign is difficult to account for satisfactorily.

(3) **The Conjunctiva.**—Sub-conjunctival *ecchymoses* are common as the result of contusion or violent fits of coughing (as in whooping-cough). Beyond a wet compress or similar application, they require no treatment. Everyone is familiar with the frequency of *foreign bodies* in the conjunctival sac, and the annoyance they produce by scratching the intensely sensitive cornea ; they are easily removed by everting the lid (generally the upper). In everting the upper lid the patient should always be directed to look well down, as comparatively large bodies often get into the upper fornix and escape observation. I have seen a large wheat grain, which had begun to sprout, removed from this situation. *Lacerations* of the conjunctiva should be carefully cleansed, and stitched if necessary. Injuries by *caustics*, *burns* by exploding powder or molten metal, and *scalds* have very often grave results, even when the cornea is untouched, and hence demand prompt and careful treatment in every instance. The most serious of these injuries is that due to the entrance of unslaked lime into the eye, a common accident amongst labourers employed in mixing mortar ; even a small particle entering the eye gives out great heat by absorbing water, and burns severely the tissues it comes in contact with, forming grey sloughy patches surrounded by inflamed areas. On the separation of the sloughs raw surfaces appear, which if opposed adhere and cause distressing deformities, such as symblepharon (adhesion of lid to globe). The eye should be first cleaned out thoroughly with milk or oil, *not* water ; many surgeons then drop in concentrated solution of sugar, as cane sugar forms an

insoluble compound with lime; I know of nothing, however, which gives greater relief or is followed by better results than castor-oil, to which cocaine may be added if the pain be great. In the subsequent treatment care should be taken to separate daily opposing raw surfaces, while inflammation is combated by cold applications and atropine. In many cases symblepharon cannot be avoided, and will require a plastic operation. Injuries of the conjunctiva by *vitriol and other acids* are frequently accompanied by corneal complications leading to nebulæ and leucomata; treatment is the same as for lime injuries, except that washing out should be done with an alkaline solution. The same treatment serves for burns or scalds.

(4) **The Cornea and Sclera.**—Slight *abrasions* are frequently caused by contusion; they speedily heal without resulting opacity under atropine and a pressure bandage. Rupture of the cornea is very rarely caused by contusion; when rupture of the globe follows very severe injury it is almost invariably in the anterior portion of the sclerotic that the rent appears (*vide infra*). *Non-penetrating wounds* of the cornea and sclerotic, if aseptic, heal quickly, leaving small cicatrices if of any depth. *Foreign bodies* embedded in the cornea should be dug out with a "spud" after previous cocainisation; when a foreign body, such as a linear chip of steel, projects into the anterior chamber it may be necessary to introduce a broad knife into the chamber to press it forward, so that it may be pulled out with fine forceps. *Burns* by heat, lime, or acids produce irritable ulcers, which must be treated by atropine, warm applications, and a pressure bandage. In cases of injury by gunpowder explosions powder grains in great numbers are often embedded in the cornea and conjunctiva, and it may be found impossible to remove all. They appear, however, to be quite inert, and rarely give rise to septic trouble.

(5) **Iris, Ciliary Body, Chorioid.**—A very frequent result of blows is *hyphæma*, or effusion of blood into the anterior chamber. Doubtless there is always some injury of the iris tissue in these cases, but it often scapes detection. The blood falls to the bottom of the

anterior chamber, where it is plainly visible ; the clot gets smaller and darker each day, and is very speedily absorbed. *Iridodialysis*, *iridoplegia*, *cycloplegia*, and *inversion of the iris* may also follow contusions. Iridodialysis, or detachment of the iris from the ciliary body, is always accompanied by (often profuse) hyphæma. It is easily recognised, a black arc—showing red with transmitted light owing to fundus reflex—being visible at the part of the periphery where the iris has been torn away ; while the pupil is altered in shape, being straightened out at the side corresponding to the iridodialysis. Sometimes we find more than one such rent ; and very rarely indeed the whole iris may be torn away, falling to the bottom of the anterior chamber. Iridoplegia, or "traumatic mydriasis," is, I believe, always due to laceration of the sphincter ; it may be accompanied by loss of power of accommodation (cycloplegia). A rare result of injury is inversion of the iris ; the iris is apparently absent at one point (traumatic coloboma), owing to a laceration the sides of which have folded back. All these lesions are permanent ; but the usual treatment of iritis should be adopted in recent cases, as traumatic iritis may supervene.

Ruptures of the chorioid are by no means infrequent after severe blows. After the dispersion of the vitreous hæmorrhages accompanying them, they are easily recognised as long linear gaps through which the white scelera shows. The ruptures usually take place a little to the temporal side of the disc, where they show as parallel crescentic whitish lines with their concavity towards the disc.

(6) **The Lens and Vitreous.**—*Dislocation* of the lens is a very common result of contusions. *Partial* dislocation is easily recognised when the edge of the lens can be seen in the undilated pupil ; the patient complains of monocular diplopia owing to one part of the pupil (usually the upper) being aphakic. On the other hand, *slight partial* dislocation is easily overlooked. The patient probably only complains of defective vision owing to the astigmatism produced. On close inspection a very pretty sign—" iridodonesis," or tremor of the iris when the eye moves, due to the want of its usual

support—is pretty constantly observed. If now the pupil be thoroughly dilated and examined with transmitted light while the eye is moved up, down, in, and out, as far as possible, the edge of the luxated lens will be seen as a black crescentic line at some part of the periphery of the pupil. The power of accommodation in the affected eye is, of course, lost. Little can be done in these cases beyond attempting to improve the vision by means of a lens empirically selected, or, if the double vision be an annoying feature, by a stenopœic ground-glass disc. In *complete* dislocation the lens falls into the anterior chamber, where it is easily seen, or is "couched" back into the vitreous, where it may be discerned as a dark body. If in the anterior chamber, it generally causes irritation sooner or later, and in any case becomes opaque; it is therefore best removed, though as the vitreous is often very fluid in these cases the operation may not be free from danger. In dislocation into the vitreous it often gives little trouble beyond that occasioned by its moving about; should symptoms of irritation arise, it must, if possible, be removed by the spoon, and in the event of failure the eye must be enucleated.

Traumatic cataract is a curious but fortunately relatively rare sequela of contusion. In most cases—personally, I think in all—the capsule of the lens is ruptured, the opacity being then produced by the entrance of aqueous or vitreous. Lawford had the opportunity of examining the enucleated eyes in two cases of traumatic cataract due to contusion, and in each case found rupture of the posterior lens capsule. Many observers, however, believe that simple "concussion" cataracts, without rupture of capsule, may develop after severe blows; and cataract has been produced experimentally by striking the eye repeatedly with a glass rod (Berlin). The first evidence of traumatic cataract is usually a small grey opacity on the anterior or posterior surface of the lens, appearing as a black dot with transmitted light; the opacity occasionally remains circumscribed, but in most cases the whole lens becomes opaque within a few days, and will then present the familiar picture of cataract, a grey opaque mass in the

pupillary area, showing up black with transmitted light. In rare instances it may happen that a traumatic cataract becomes absorbed; nearly always, however, it becomes shrivelled and calcareous. When it is accompanied, as it often is, by other contusion injuries of the iris, ciliary body, or uvea, the resulting inflammation may cause it to adhere to the iris, or even provoke such general inflammation of the eye as to destroy it completely. On the whole the safest course—a course which is imperative if there be increased tension—is to remove the lens as speedily as possible, though in cases in which it is adherent to surrounding parts the operation may be attended with considerable difficulty.

Hæmorrhages into the *vitreous*, seen as dark moving masses by transmitted light, frequently follow contusion; if not absorbed they may undergo conversion into connective tissue, which may be vascularised by vessels running from the retina, forming what is known as *retinitis proliferans*. Appearances indistinguishable from retinitis proliferans are, however, sometimes congenital; and the fact that large recurring *sub-hyaloid* hæmorrhages are fairly common without obvious cause in adolescents (I have seen several cases in young soldiers) should also be remembered, as it may prove of medico-legal importance.

(7) **The Retina and Optic Nerve.**—A general haziness with slight swelling of the retina, known as *commotio retinæ*, is frequently observed after injuries; it is probably due to œdema, and usually subsides quickly. *Atrophic* changes in the macular region, resembling those seen in high myopia, are occasionally seen as a late sequela. A far from infrequent, and very serious, consequence of contusions is *detachment of the retina;* a lesion also seen in high myopia, and occasionally as an apparently spontaneous occurrence in healthy eyes (*vide* Chapter IV.). As a matter of convenience, and also because I believe many of the myopic and "idiopathic" cases are due to unnoticed injury, all three varieties of detachment will be considered here. On looking at the eye with transmitted light, a greyish veil on which the vessels appear as black lines will be seen floating up from below in the red pupil, if the detachment be

partial, or occupying the whole pupillary area if complete. On direct examination, part or the whole of the retina, generally moving, will be found much nearer the observer's eye than the disc, so that to see it distinctly, and focus the vessels on it properly, + lenses, varying from + 3 or 4 near the disc to perhaps + 12 at the periphery, will be required ; a rent in the detachment is frequently seen. Cases of detached retina are thus easily enough diagnosed when a large part or the whole of the retina has been dislocated, and when large vitreous opacities are not present ; not so, however, when the detachment is very small and limited, as in cases where little ridges appear on the retina, or when it is accompanied by large vitreous hæmorrhages. The detachment does not always move ; in such cases a guarded diagnosis should be given until the case has been watched for some time, as the retina may be detached by a tumour, such as glioma or sarcoma, necessitating early removal of the eye, and involving a very grave prognosis. Occasionally, very rarely however, the chorioid is detached with the retina, through which its tesselated surface is recognised. Disturbance of vision is great in detached retina, the patient complaining of "a veil over the sight" : the field of vision presents a large scotoma corresponding to the detachment. As a rule the upper part of the retina is the portion first detached, but by gravitation of the subretinal fluid the detachment extends rapidly to the lower part, the upper probably adhering again.

The mechanism by which the retina is detached as the result of a violent blow is not difficult to understand ; but the exact cause of myopic and idiopathic detachments when injury can be excluded is still unsettled, though many more or less plausible theories have been advanced. In some cases it seems certain that the detachment is caused by the traction of adhesions between the vitreous and retina. In the majority, however, recent experiments seem to show that the "diffusion theory" is the correct one.

Sinclair[1] produced detachment in rabbits' eyes by the

[1] *Journal of Pathology*, June, 1901.

post-retinal injection of salt solution, and also of a highly albuminous fluid (blood-plasma); in the latter case the detachment progressed after injection owing to continued accumulation of fluid diffused from the vitreous. The vitreous fluid is almost entirely composed of water (98 per cent.), while in inflamed conditions post-retinal transudation of a highly albuminous fluid takes place. Looking at the retina as a dialysing membrane, when there is an accumulation of inflammatory exudation behind the attachment of the retina this will tend to increase by the process of diffusion of the vitreous fluid through the retina; cases where the extension of detachment occurs rapidly without increase of intra-ocular tension can be accounted for in no other way. The fact that changes in the vitreous often take place in eyes in which detachment subsequently occurs cannot be taken as any proof that these changes cause detachment of the retina. The truth would appear to be that both conditions are brought about by uveal inflammation, and are therefore often found together.

The prognosis of detached retina is extremely bad. Many treatments have been suggested and tried, but all are unsatisfactory. Cases have been recorded in which the retina has been found re-attached after prolonged rest in bed in one position; but when the after history of these patients has been followed up one generally finds that the detachment recurred. The prospect of cure by this means is so very slender that it seems hardly justifiable to make a patient undergo a very trying ordeal when the result is so problematical. Evacuation of the sub-retinal fluid through a sclerotic incision is another procedure which has its advocates. I have never seen the slightest good result from it. Sub-conjunctival injections of chloride of sodium solution have also been tried and found wanting. In myopic and idiopathic cases absolute rest for the eyes and the use of dark glasses should be ordered, as there is always a probability that the other eye may be affected.

The optic nerve may be contused, compressed by effusion, or actually torn across by fractures traversing the optic foramen: but such cases are happily very rare.

Blindness is the immediate result, followed in process of time by a dead-white atrophy of the disc.

Lightning Injuries.—Various eye lesions have been caused by, or ascribed to, lightning stroke. *Burns* of the lids, conjunctiva, or cornea due to this cause differ but little from other burns of the eye. Nine cases of *cataract* following lightning stroke have been recorded. Spasms or paralysis of ocular muscles, optic atrophy, neuro retinitis, and retinal hæmorrhages have also been described.[1]

I saw two cases of injury to the deeper parts of the eye following lightning stroke during the South African Campaign. Of these the second case is, I believe, unique. Both were recorded by me in the "Transactions of the Ophthalmological Society" for 1901.

The first was in the person of a brother officer whose right eye was rendered blind by thrombosis of the central vein. His tent had been struck by lightning while he was sitting in it; at the time he felt a sharp, painful thrill run down the right side of his body from head to foot, but did not notice any visual trouble till some weeks later, when he discovered accidentally that his right eye was blind. I saw him two months after the accident at Cape Town; he had then large macular hæmorrhages of some standing, and other signs of thrombosis. The diagnosis was somewhat doubtful, as he had had an attack of jaundice about a month before the lightning stroke. He was invalided to England, when he consulted Mr. Nettleship and Mr. Treacher Collins. The former thought the lesions were probably due to lightning, while the latter seemed inclined to attribute them to the jaundice.

The second case occurred in an Army telegraphist, known to have been emmetropic previous to the injury. He was working a telegraphic instrument in a small hut during a storm, when a flash of lightning crumpled up the instrument into a shapeless mass, and "knocked him senseless to the ground." He recovered consciousness in a short time, and then found he was blind. I saw him a few days later, and found he had a retinal

[1] *Vide* an excellent paper on the subject by Rohmer, *Arch. d'Ophtal.*, April, 1895.

detachment in each eye—complete in one, nearly complete in the other. In this case the connection between the lightning stroke and the eye lesion seems clear.

Doubtless lightning acts on the eyes in several different ways—by heat, light, concussion, and possibly some obscure physico-chemical action. Rohmer draws attention to the analogy between the lesions produced by lightning and those due to electric light. Spasms and paralyses of the ocular muscles are evidently due to the direct effect of electricity on the nerves. Silex attributes lens opacities to coagulation of the albuminous fluid by the catalytic action of the electric discharge. In Hess's experiments on rabbits cataracts were shown to be caused by the destruction of the intra-capsular epithelium. If we admit the possibility of contusion producing simple "concussion cataract," lightning stroke might conceivably act in the same way.

(*b*) CONTUSION WITH RUPTURE OF GLOBE.

Rupture of the globe may follow a very violent blow; it is a very grave injury, and generally means complete destruction of the eye. The most frequent site of rupture is in the sclerotic just behind the upper and inner margin of the cornea; the cornea itself generally escapes owing to its elasticity. The retina is almost certain to be detached, while iris, ciliary body, lens, and vitreous may each or all prolapse through the rent; the prolapse being sub-conjunctival in cases in which the conjunctiva is untorn. Should there be no prolapse, there is just a chance that the case may do well and recover with a certain amount of vision.

In cases of ruptured globe in which there seems to be a possibility of saving the eye, prolapses may be snipped off and the lips of the wound brought together by sutures through the episcleral tissue and conjunctiva. If, however, the rupture be extensive, it is best to enucleate at once, as, even if it heals, the eye will be useless and a source of danger to the other.

(*c*) PENETRATING WOUNDS.

Penetrating eye-wounds are always serious injuries, more especially when complicated by the retention of

the penetrating agent. The danger of the introduction of septic matter into the eye through incised and punctured wounds is very great, greater probably than in other parts, as there is seldom free bleeding externally. Cases of foreign bodies in the eye probably demand more judgment and skill than any other class of cases in ophthalmic surgery, for the wound of entrance is often so small as to leave no appreciable cicatrix behind, while till the introduction of X-rays it was quite possible to overlook the presence of a small body in the eye in examining by ordinary methods.

Rifle or shell wounds of the eye are very grave lesions indeed, partly *per se*, and partly because they are almost invariably accompanied by injury of the orbit and skull generally. The prognosis as regards life in severe gunshot wounds of the orbits is not so bad as might be expected. Twenty-five examples of recovery after evulsion of both eyes by shot traversing the orbit are recorded in the "Medical and Surgical History of the War of the Rebellion." The following case of bullet wound (Mauser) which I saw in the South African War is of interest in this connection :—

"Captain T., Gordon Highlanders, was wounded in an action about twenty miles away, and brought in here (Bloemfontein) last night. This case was the worst I have yet seen. The bullet entered his left temple, crossed the left orbit—reducing the eye to pulp, passed through the ethmoid cells, through the frontal lobe of the brain, thence out through the upper and inner angle of the right orbit—reducing the right eye also to pulp : brain substance was exuding through the wound of exit. Another bullet had passed through the right groin, in dangerous proximity to the femoral artery. The remains of the eyes were removed, and a gauge drain inserted in the orbital bullet track, the femoral wound being treated by a simple antiseptic pad over entrance and exit. He recovered without the slightest bad symptom."[1]

Of the 825 cases of gunshot wounds destroying the sight of one eye recorded during the American Civil War, 91 were attacked by sympathetic ophthalmia in

[1] Yarr: "Medical Notes from South Africa." Bale, Sons and Danielsson, 1901.

the remaining eye (11 per cent.). No specific examples of eye injuries from stones or other hard bodies thrown up by shells or by the impact of large projectiles on masonry seem to have been noted in that campaign. Injuries due to the intrusion of fragments of percussion caps into the eye numbered only 19 : from the scanty details of these the following facts may be gleaned :—

1. Men returning to duty had non-penetrating injuries of the globe.
2. The eye was lost when the foreign body entered the posterior chamber.
3. The only recoveries after entry into the anterior chamber were in cases where the foreign body was at once extracted.

The editor of the "History" adds that "a general survey instructs that all foreign bodies lodged in the globe should be extracted at all hazards."

Cohn (quoted by Gruening) described 31 cases of gunshot wounds of the eye observed by him in the Franco-German War of 1870-71, and enumerated no less than seventy different pathological conditions found in the injured eyes. The chief lesions observed were wounds of globe with and without retention of foreign body, iridodialysis, detachment of retina, rupture of chorioid, paresis of internal rectus. In 2 cases the projectile struck the brain, in 4 the cranial bones, in 9 the face, and in 16 the eye directly.

Penetrating wounds of the eye by small shot generally involve loss of the eye. A shot has been known to pass half round the eye beneath the conjunctiva without penetrating the globe.

Penetrating wounds of the cornea are almost certain to be complicated by prolapse of the iris ; and, if deep enough, by traumatic cataract, or dislocation or even extrusion of the lens. Wounds implicating both cornea and sclerotic are always serious, owing to the risk of sympathetic ophthalmia following injuries in this region. In such cases the eye may be lost at once owing to extensive traumatism, or subsequently reveal traumatic inflammation. All that can be done in penetrating wounds of the globe in which there is no evidence of retained foreign body is to irrigate the eye

thoroughly with antiseptic solution, and to snip off any protrusions of iris, cauterising the stump if sepsis be probable; a gaping sclerotic wound should be brought together by stitches through the conjunctiva and episclera. Should symptoms of panophthalmitis develop, the eye must be excised. The whole subject of penetrating wounds must, however, be considered in connection with that of sympathetic ophthalmia (*vide infra*).

Serous cyst of the iris is an interesting but rare sequela of penetrating wounds; such cysts have been proved by experiments to be due to implantation by the penetrating body of epithelium brought from without; this epithelium grows until its cells are pushed apart by fluid and converted into a cyst wall. Cysts grow slowly until they partly fill the anterior chamber, set up increased tension, and so cause blindness. Early removal by an incision at the corneal margin, through which the cyst, with the portion of iris from which it grows, is pulled out and cut off, is indicated.

Traumatic cataract is occasionally absorbed, lens substance being gradually extruded through the torn capsule, and absorbed piecemeal. A cataractous or dislocated lens causing evident irritation should be extracted.

Injuries of the chorioid and retina generally involve detachment of both and sepsis of the eye; cases, however, sometimes occur in which nothing is left except pigment disturbance in the fundus at the site of penetration.

Penetrating Wounds with Retention of Foreign Body.—A golden rule of ophthalmic surgery is to *suspect* a foreign body in all cases of penetrating wounds. In many foreign body cases the objective symptoms are so slight that patients do not consult a surgeon until some time after the accident, and by that period the scar of the entrance wound may be difficult to distinguish; hence the *history* should always be closely investigated. The bodies most commonly found in the eye are chips of steel, lead, copper, and bits of stone or glass. Of these copper is the most dangerous, as it speedily sets up purulent inflammation by chemical action. To obtain entrance

into the eye a body must evidently be both small and swiftly moving; hence in many instances the wound closes at once without prolapse of iris or escape of fluid. The wound of entrance is generally situated in some part of the cornea, or in the corneo-scleral margin.

The foreign body after entering may fall to the bottom of the anterior chamber, or pass into the lens either through the iris or in the pupillary area, or go right through the lens into the vitreous, remaining there or sticking into the tunics of the eye.

In the anterior chamber it may resemble a small hyphæma owing to being covered with blood clot; it is easily removed through a marginal incision by a magnet or forceps.

If it stick in the lens, it is advisable to postpone active interference till the resulting opacity has extended throughout the lens, and then to extract the cataract; unless increased tension or evidences of inflammation necessitate earlier action.

Foreign bodies in the vitreous—even in cases in which the lens is clear and there are no vitreous hæmorrhages — are often difficult to detect. Sometimes a grey or reddish streak extending back from the wound of entrance shows the direction taken; but this may be absent. The body usually falls to the bottom of the vitreous, where it becomes covered with exudate; sometimes it moves freely about with the eye movements; sometimes, again, it is embedded in the retina, and may be too far forward to be seen. Looked at with the ophthalmoscope soon after the accident, it generally appears as a dark or black body; later it often presents a peculiar white glistening appearance, like a tiny electric globe. Cases in which a foreign body is suspected, but cannot be found, should always be X-rayed—if possible by an *ophthalmic* X-ray expert, as he can localise the exact position of the body in the eye, or in the orbit, for it may have passed right through the globe. X-ray experts will find Mr. Mackenzie Davidson's method of localising foreign bodies in the eye or orbit by far the best; the details are too lengthy to transcribe here, but they will be found most lucidly set forth in an admirable paper by

that gentleman in the 1898 volume of the "Transactions of the Ophthalmological Society."

Haab's enormous magnet, which I have seen in use at Zurich, and which was shown before the Ophthalmological Society by Mr. Bickerton in 1899, is useful both in localising and removing foreign bodies, but its great size and weight render its general use in military practice out of the question. A patient suspected of having a foreign body in his eye is placed with the eye close to the instrument (which is worked by a dynamo or current from the street); should a metallic foreign body be present, he will at once complain of acute pain, owing to the traction on the metal. In many cases the body can be drawn into the anterior chamber, whence it is easily

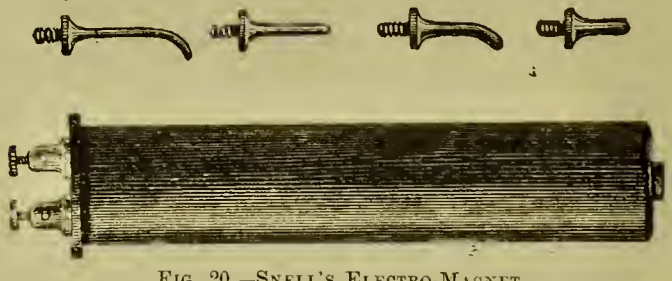


FIG. 20.—SNELL'S ELECTRO-MAGNET.

removed, or even through the wound of entrance. Mr. Holmes Spicer, of Moorfields, has quite recently published a remarkable series of cases in which Haab's magnet was employed for the detection and removal of foreign bodies.[1]

The "sideroscope" shows the presence of steel or iron in the eye by the deflection of a magnetic needle shown on a scale which has to be read by an astronomical telescope. I have never seen this instrument.

There is only one *treatment* for foreign bodies in the eye, and that is to extract them as soon as possible. Though a foreign body *may* become encapsuled in exudate, and give no trouble for years perhaps, it is almost

[1] *British Medical Journal*, Jan 18th, 1902.

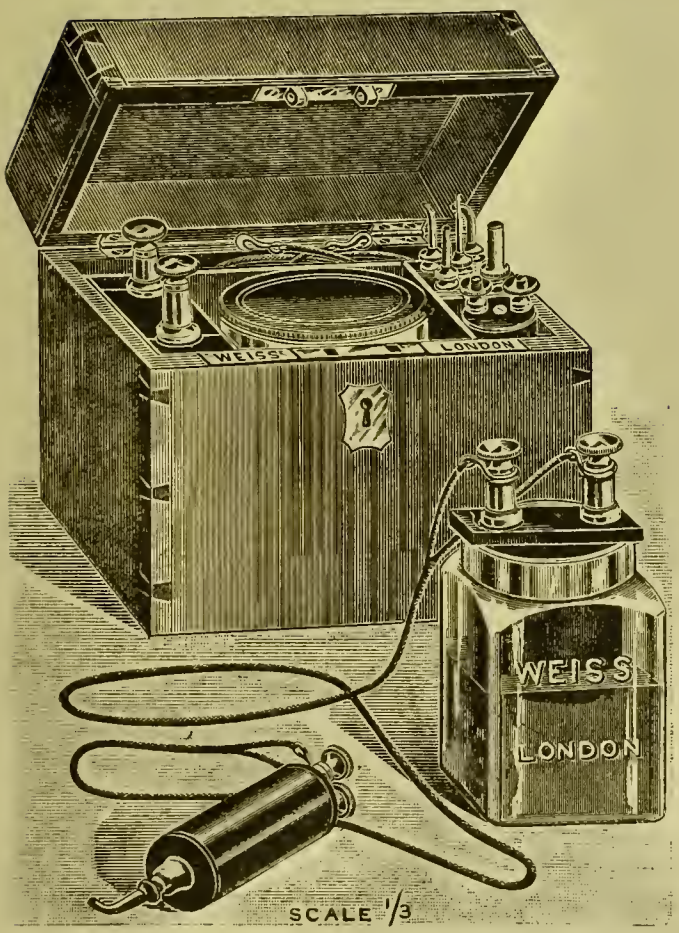


Fig 21.—Weiss's Electro-Magnet, with Battery.

certain to excite inflammation sooner or later (*vide* next chapter). Chips of copper are the most dangerous foreign bodies, as they are most certain to give rise to destructive inflammation; fortunately they are rarely seen nowadays, since brass percussion caps are now only seen in toy pistols.

Having localised the foreign body by the ophthalmoscope, X-rays, or other means, an incision must be made through the sclerotic as near it as possible : to do this it may be necessary to cut the tendon of a rectus muscle and forcibly rotate the globe. A general anæsthetic is necessary. The incision should be from before backwards, parallel to and between the recti. If the body be non-magnetisable, an endeavour must now be made to catch it with forceps while it is kept in view by the ophthalmoscope. If magnetisable, an electro-magnet should be employed (see figures).

Weiss's electro-magnet, fitted in a mahogany case with a bichromate battery, is a very convenient one. A needle of the most suitable size and shape is fitted on the magnet and introduced ; when caught by it, the foreign body must be carefully manœuvred out through the operation wound. Of course, vitreous is lost during the operation, but it is surprising how quickly it is replaced. Needless to say, the strictest antiseptic precautions are necessary.

"Siderosis" is an expression used to describe certain degenerative changes in the ciliary body, retina, and lens capsule following the intrusion of iron into the eye, probably due to its solution. Van Hippel has recorded a case in which the whole retina was in this way converted into pigmented tissue.

CHAPTER XIII.

SYMPATHETIC OPHTHALMIA.

Sympathetic ophthalmia is a plastic uveitis in one eye, excited by a *traumatic* irido-cyclitis in the other.

It is *not* excited by anterior staphyloma following perforated ulcer, gonorrhœal ophthalmia, etc., by panophthalmitis, by absolute glaucoma, by intra-ocular tumour, or by non-penetrating injuries. Exceptions are so exceedingly rare that they should be looked on as ophthalmological curiosities, not as guides to practice.

A certain period of time—seldom less than three, or more than twelve, weeks—elapses between the injury to the exciting eye and the commencement of inflammation in the sympathising eye.

It is not a common disease.

I put these propositions in the forefront of this chapter in order to clear the air *ab initio*, for they all bear on points on which great misconception prevails.

CAUSATION AND PATHOGENY.

The cause of the exciting irido-cyclitis is, it may be safely said invariably, a *penetrating wound*, with or without retention of a foreign body. Cases of sympathetic ophthalmia following contusion without rupture, or with sub-conjunctival rupture, have been reported, but few, if any, bear close investigation. It may, then, be accepted as certain that communication between the external air and the interior of the eye is a necessary factor in the origin of the exciting irido-cyclitis. Of all wounds producing exciting irido-cyclitis, those implicating the *ciliary* region are the most likely to set up that particular form of the disease which excites sympathetic ophthalmia in the other eye ; and there is a general consensus of opinion that wounds of this region are

especially dangerous when accompanied by prolapse of iris or ciliary body. For this reason sympathetic ophthalmia has been known to follow cataract extraction. Traumatic irido-cyclitis due to a retained foreign body is practically certain to excite sympathetic ophthalmia.

The *pathogeny* of sympathetic ophthalmia is one of the most vexed questions in ophthalmology. A mass of literature has been produced and hosts of experiments performed by able investigators with a view to furnishing a satisfactory answer to the question, "How does the inflammation travel from the exciting to the sympathising eye?" Yet it must be confessed that we have not yet found a certain solution of the problem.

The earliest theory, propounded by Mackenzie in 1854, was that inflammation from the exciting eye travelled along the corresponding optic nerve to the chiasma, and thence by the other optic nerve to the sympathising eye. Later, Müller endeavoured to show that the inflammation was transferred along the ciliary nerves; a theory which prevailed for many years. Within recent times the subject has been taken up and investigated scientifically, notably by Deutschmann, Gifford, and Randolph. with the result that the old view enunciated by Mackenzie, expressed in more modern form, is now that most generally adopted. Deutschmann injected the spores of "aspergillus fumigatus" in some experiments, croton oil in others, into the vitreous of a rabbit's eye, with the result that inflammation appeared in the other eye; and he was able to demonstrate an infiltration passing from the exciting eye along its optic nerve, across the chiasma, and down the other optic nerve to the sympathising eye. Gifford performed similar experiments on rabbits, using pyogenic organisms as the excitant, but was unsuccessful in producing sympathetic ophthalmia; he then tried the anthrax bacillus, but only succeeded in three out of twenty-five cases. Randolph failed with the staphylococcus aureus, experimenting on dogs as well as rabbits. Notwithstanding these failures, it seems almost certain that the disease is transferred from one eye to the other *viâ* optic nerves and chiasma; and extremely probable that it is "an inflammation due to micro-organisms, and propagated by them to the

sympathising eye by direct continuity through the optic nerves and chiasma, as erysipelas extends over the skin" (Swanzy).

DIAGNOSIS AND SYMPTOMS.

It must always be remembered that sympathetic ophthalmia is, relatively to the number of cases of penetrating wound of the eye, an infrequent disease. On the other hand *sympathetic irritation* in one eye, as shown by discomfort, slight conjunctival injection, and asthenopia ("weak sight") complicating trauma in the other, is quite common. At one time it was believed that sympathetic ophthalmia was generally preceded by a prodromal stage of irritation. We now know, however, that in most cases there are no prodromal symptoms, and the disease is already under way when first seen. The moral is that patients who have suffered an eye trauma likely to cause sympathetic ophthalmia should be seen at frequent intervals within the first few months after receipt of the injury.

In a suspected case the first sign to be looked for is a *keratitis punctata*, for the disease begins as a serous irido-cyclitis. In a typical case the patient presents himself complaining of a "haze over the sight": the visual acuity is reduced, the tension probably unaltered; there may be slight pericorneal injection; the iris is dull, the pupil sluggish and immobile, the anterior chamber deep; spots of keratitis punctata appear on the back of the cornea; the fundus looks hazy, the disc being pink and blurred from clouding of the media; rarely there is some slight neuritis. From this on the progress of the disease is slow and insidious, with frequent periods of quiescence. The haziness of sight increases; slight pain may now be complained of; the tension becomes *plus* to a varying extent, while the anterior chamber gradually gets shallower and shallower; the iritis assumes a plastic form, the iris becoming sodden and rigid; lymph is deposited in the pupil, perhaps entirely filling it up (occlusio pupillæ); infiltration of the ciliary body and chorioid follow, and the whole nutrition of the eye is impaired. After a time the tension of the eye becomes (sometimes suddenly)

minus, and ultimately the eye is converted into a useless shrunken globe (phthisis bulbi). In a few rare instances the disease process has been known to stop before the iritis assumed the plastic form; but in the vast majority the progress of events to more or less complete blindness is sure, though it may be slow.

Cases have been recorded in which a *neuro-retinitis* alone in one eye followed trauma in the other; but a careful perusal of the published notes of these cases leads one to the belief that this neuro-retinitis was not of the nature of sympathetic ophthalmia. On the whole one is justified in the present state of our knowledge in asserting that irido-ciliary lesions form an essential and never absent manifestation of sympathetic ophthalmia.

Deafness, from implication of the auditory nerve, is a rare complication.

Sympathetic ophthalmia as a rule appears at some time between the third and twelfth week after the injury, usually when the exciting irido-cyclitis is at its height. There are, however, some few authentic cases in which the disease appeared earlier than the third week, in two at least as early as the fourteenth day after the trauma. Cases in which it appears later than the twelfth week are more frequent. I have seen three such at Moorfields—in one after a lapse of five months, in another nine, and in a third two years; in all these cases, however, there had been a recrudescence of the exciting irido-cyclitis before inflammatory symptoms manifested themselves in the sympathising eye. Knapp has described a case in which sympathetic disease appeared forty-five years after injury!

As regards *prognosis* when the disease is established, complete recovery is very rare. Randolph was only able to collect nineteen well established cases, all under the age of forty, and many of them children. Youth is apparently a favourable factor.

TREATMENT.

"The prophylactic treatment naturally plays a most important *rôle* in sympathetic ophthalmia, and of course the only certain prophylaxis is the enucleation of the injured eye. . . . By certain prophylaxis I mean

relatively certain, for we can never assert positively that sympathetic ophthalmia will be surely averted if the injured eye be enucleated, nor, indeed, can the assertion be made that in any case where the other eye is perfectly sound it will become diseased unless the injured eye be enucleated." (Randolph.)

Obviously a certain preventative of sympathetic ophthalmia would be the enucleation, immediately after the injury, of an eye effected by a trauma likely to give rise to sympathetic ophthalmia in the other ; and this is undoubtedly a correct procedure in cases of intrusion of foreign bodies which we are unable to extract. But we have only to consider the relative infrequency of sympathetic ophthalmia to realise that it would not be fair to a patient to remove an injured eye (except in the case of retained foreign body) if he had a fair amount of vision in it.

Mr. Swanzy lays down some excellent axioms on the subject in his "Handbook of Diseases of the Eye." Every case must, however, be carefully thought out on its own merits ; though it is well to approach the consideration of an individual case with some general classification of eye traumas, with reference to prophylaxis of sympathetic ophthalmia, in one's mind. The following is a nucleus, at all events, of such a classification.

1. Cases in which an Injured Eye should be Removed.

(*a*) When it contains a *metallic* foreign body which we cannot extract.

(*b*) When its vision is so bad that it is useless, and there is persistent sympathetic irritation in the other.

(*c*) When it is blind and unsightly, and the injury has been such as to render sympathetic ophthalmia a probable sequela.

(*d*) When, being blind or nearly blind, it has already excited sympathetic ophthalmia in the other eye ; for its removal *may* exert a favourable influence on the sympathising eye.

2. Cases in which it is Safer to Remove an Injured Eye.

(*a*) Sometimes a non-metallic foreign body remains encapsulated or embedded without causing any trouble; it may perhaps be permissible in such cases to leave the eye, if its vision be good and the patient lives within reach of a specialist. On the whole, however, it is better to enucleate.

(*b*) When, though its vision is not completely destroyed, the eye is injured in such a way as to render sympathetic ophthalmia probable, in a patient who has to live out of reach of skilled aid.

3. Cases in which an Injured Eye Should Not be Removed.

(*a*) When its vision is good or fair, and sympathetic ophthalmia has already attacked the other eye. For, as enucleation of the injured eye *may* have no effect in stopping the progress of the sympathetic ophthalmia, we have no right to deprive the patient of what might prove to be his only seeing eye.

(*b*) We are not justified in removing a seeing injured eye merely because sympathetic *irritation* appears in the other.

As the passage of the disease from the exciting to the sympathising eye takes some time, it is quite possible for disease to break out in the latter a considerable time after the removal of the former. Mr. Holmes Spicer ("Transactions of Ophthalmological Society," 1899) has recorded a case of onset of sympathetic ophthalmia a fortnight after removal of the exciting eye. We must therefore give a guarded, though hopeful, prognosis in case of enucleation, even when the remaining eye appears absolutely sound.

Many surgeons prefer evisceration by Mules' operation to enucleation, but I am sure the latter is the safer in cases of threatened sympathetic ophthalmia, though doubtless Mules' operation is preferable when excision for some other reason is required.

It is to be feared that treatment has but little effect in arresting the progress of sympathetic ophthalmia

once it is established in the sympathising eye ; too often what looks like an arrest is merely an intermission. Hot applications, atropine, and complete rest in a darkened room relieve symptoms, and form the usual procedure in these cases ; on the appearance of increased tension the atropine must be stopped. Cocaine is useful when pain is a prominent symptom. Abadie believes he has succeeded in arresting the disease by the injection into the eye of a drop of 1 in 1,000 corrosive sublimate solution, combined with cauterisation of the exciting eye at the site of the injury.

Mr. Swanzy lays down the "golden rule" that no operation for the formation of an artificial pupil should be attempted till at least twelve to eighteen months after the onset of the disease, as operative interference at an earlier date will only aggravate or rekindle inflammation. Iridectomy is always a difficult, and sometimes an impossible, operation in sympathetic ophthalmia, for the rigid, sodden iris tears when caught in the forceps ; and even when a satisfactory gap has been made the improvement in vision may be *nil*, owing to the lens being covered by lymph. Critchett's operation of discission of the lens has succeeded in restoring sight in a few cases. He introduced two needles into the same part of the lens from opposite sides of the cornea, and then, by bringing the ends together, separated the points so as to make a wide tear. The operation must be repeated as often as necessary until the absorption of the lens is complete.

CHAPTER XIV.

GLAUCOMA.—CATARACT.

THOUGH glaucoma, even in its secondary form, is not very often seen in military practice, it is desirable to give a brief sketch of the pathology, diagnosis, and treatment of this condition, as there is no eye disease in which errors of diagnosis are more frequent or more disastrous.

GLAUCOMA.

Glaucoma may be defined as a state of increased tension of the eye due to obstruction of the means of exit of intra-ocular fluid. It is known as *primary* when it supervenes without any previous (discoverable) lesion ; *secondary* when it is evidently due to antecedent disease.

Pathogeny.—The aqueous humour in the normal eye is maintained in a state of continuous though slow circulation. Fluid pours into it from the secreting cells of the ciliary body (first minutely described by Mr. Treacher Collins), and drains away from it at the angle of the anterior chamber—the "filtration angle"—into the canal of Schlemm, whence it finds its way into the veins ; and it is only by a proper balance being maintained between these processes of secretion and excretion that the intra-ocular tension is maintained within normal limits. It is evident, then, that if the outflow be checked or obstructed in any way increased tension must follow ; and this is exactly what happens in glaucoma. "In all forms of glaucoma, without exception, changes likely to obstruct the escape of fluid at the filtration angle have been found. Moreover, in a considerable number of eyes blinded by glaucoma the permeability of the filtration angle has been tested by injection of fluid into the anterior chamber, and in

every instance a great impairment of filtration has been found" (Priestley Smith).

Primary glaucoma is a disease of middle and old age, being seldom seen before forty-five; it is nearly always bilateral, though both eyes are rarely attacked at the same time. Women are more liable to attack than men, the ratio being six to five (Priestley Smith). Hypermetropes are predisposed to it; high myopes may be considered immune. Acute mental depression seems to be a frequent exciting cause; from the out-patient department at Moorfields I carried away the impression that *women recently widowed* are specially liable to glaucoma. Exposure to cold, over eating, fatigue, disturbance of circulation from any cause, may also act as excitants in predisposed persons. Anatomically the immediate cause is narrowing or closing of the filtration angle set up by congestion of the ciliary body in an eye (such as a hypermetropic eye) in which the angle is smaller and the lens nearer to other parts than usual.

The danger of mydriatics in eyes predisposed to glaucoma, or during the "glaucoma age," is due to the fact that the dilated iris is necessarily thicker and more wrinkled than when undilated, and may therefore close up a very narrow filtration angle. Though mydriatics are in this way a real danger, and most, if not all, ophthalmic surgeons have seen cases in which atropine has had the unfortunate effect of exciting an attack of glaucoma in a predisposed eye; still, on the whole, the danger has been exaggerated, and such cases are not so frequent as generally supposed.

The chief causes of *secondary* glaucoma are annular posterior synechiæ; perforating wounds or ulcers of cornea with prolapse of iris (anterior synechiæ); cataract operations in which some part of iris or lens capsule has been caught in the scar; wounds of the lens or rupture of its capsule, causing it to swell suddenly; dislocation of lens; exudation into aqueous or vitreous; intra-ocular hæmorrhages. All these are conditions tending to block or obliterate the filtration angle. Serous cyclitis is also a frequent cause; here, however, the anterior chamber (and filtration angle) is deeper than usual, and the anatomical cause of the increased tension

seems to be that "the inflamed ciliary body pours into the aqueous chamber a morbid albuminous fluid which escapes from the eye with greater difficulty than the normal secretion" (Priestley Smith).

Symptoms.—Most cases of primary glaucoma are preceded by a *prodromal* stage which may last months or years. During this stage the patient notices that his power of accommodation is going; if he wears presbyopic glasses, he is constantly changing them for stronger and stronger ones. At frequent intervals attacks of headache, accompanied by dimness of sight and the appearance of rainbow rings round lights, come on. The casual observer probably sees nothing wrong in the affected eye during these attacks; but close examination will show a slight narrowing of the anterior chamber, a certain sluggishness of the pupil, some slight ciliary injection, and a very faint steaminess or haze of the cornea. To the last-named sign is due the appearance of rainbow rings round lights; one gets the same effect by looking at a lamp through a frosty window pane. It is a remarkable fact that sleep—even a short doze—cuts short these prodromal attacks. In the intervals between attacks vision is apparently normal.

After this stage has lasted a variable time, an attack of acute glaucoma suddenly supervenes, usually without any warning. The symptom-complex of an *acute attack* includes the following: Pains radiating from the eye all over one side of the face; vomiting; tenderness and increased tension (+ 1, + 2, or even + 3) of the eye; conjunctiva congested, cornea dull and steamy, anterior chamber shallow, pupil dilated and immobile with a green reflex (γλαῦκος = green); vision much reduced, probably to hand movement; examination of fundus impossible owing to steaminess of cornea. In very bad cases (glaucoma "fulminans") the eye becomes absolutely blind within a few hours. In most cases, however, this acute attack passes off within a few days, leaving the cornea almost, perhaps quite, clear; the anterior chamber shallow; pupil sluggish or immobile; tension noticeably *plus*; vision variably reduced; field contracted, most markedly on the *nasal* side (for the *temporal* side of the retina is the side most affected by

disease, as will be evident on examining the ophthalmoscopic picture).

The period of quiescence is followed by another attack, and that by others in quicker and quicker succession : after a time the eye becomes quite blind, of stony hardness, with widely dilated pupil and very shallow anterior chamber (*absolute* glaucoma) ; it may continue in this state for months or years, giving rise to frequent attacks of agonising pain. Finally it shrivels up and atrophies.

Sooner or later in the course of glaucoma—after the first attack if the prodromal stage has been lengthy—the

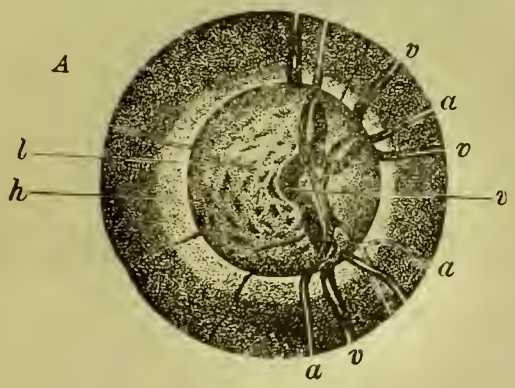


FIG. 22.—GLAUCOMATOUS EXCAVATION OF THE OPTIC NERVE. MAGNIFIED 14 BY 1. (FUCHS.)

OPHTHALMOSCOPIC PICTURE OF THE PAPILLA.—The papilla is bounded by a sharp, overhanging edge, at which the arteries (*a*) and the veins (*v*) of the retina appear to stop with their ends bent over the edge. This is due to the fact that their continuation on the floor of the excavation is often displaced somewhat laterally as compared with the portion situated in the retina; moreover, the vessels within the excavation are seen, but indistinctly. In the outer half of the excavation are seen the grey dots of the lamina cribrosa. The zone (*h*) of the fundus, adjoining the papilla, is decolorised (halo glaucomatosus).

characteristic ophthalmoscopic picture of glaucoma will be seen. The most conspicuous feature of this picture is the *cupping* of the disc, due to the pushing back of the weakest part of the sclera—the lamina cribrosa, including nerve fibres going through it—by the

increasing intra-ocular tension. At first the cupping or excavation is partial, on the temporal side : the vessels are seen *bending* down into the cup from the margin, and (in direct examination) a concave lens is required to see the bottom of the cup clearly ; at the bottom the lamina cribrosa appears as a bluish grey lattice-work. At a later stage the whole disc (or more probably three-fourths of it, for a small portion at the nasal side generally remains unaffected) becomes deeply cupped ;

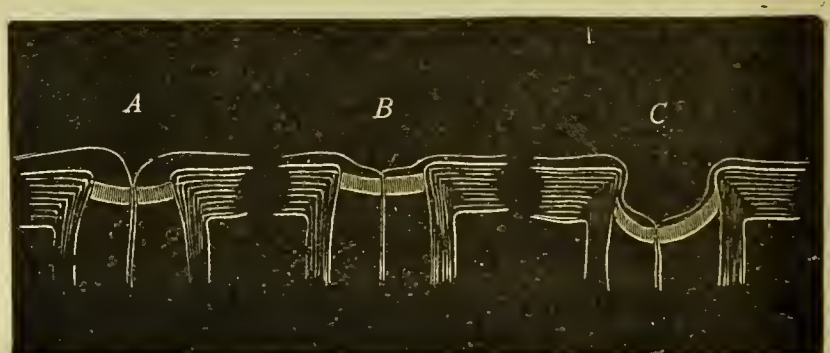


FIG. 23.—THE THREE KINDS OF EXCAVATION OF THE OPTIC NERVE.—SCHEMATIC. (FUCHS.)

A. PHYSIOLOGICAL EXCAVATION.—Funnel-shaped, partial, with normal lamina cribrosa.
B. ATROPHIC EXCAVATION.—Bowl-shaped, total, with normal lamina cribrosa.
C. GLAUCOMATOUS EXCAVATION.—Ampulliform, total, with the lamina cribrosa bulged out posteriorly.

the vessels dip sharply from the edge into the depression, or may actually bend underneath the overhanging margin so as to appear interrupted (*vide* figure); the whole disc is grey, and the lamina cribrosa distinctly visible ; round the disc is a faint whitish halo due to commencing chorioidal atrophy (the "glaucomatous halo").

The cupping due to glaucoma must be carefully distinguished from physiological or atrophic cupping. The physiological cup, though frequently large, is easily recognised as such by the fact that it never reaches

the margin of the disc ; the lamina cribrosa is really in its natural position, and the appearance of cupping is due to the optic nerve fibres spreading out behind the level of the retina. The atrophic cup does extend to the margin, but is shallow, and its sides slope ; in this cup, too, the position of the lamina cribrosa is unaltered, but the nerve fibres passing through it being atrophied the whole surface of the disc is lowered. It is, I am bound to admit, in many instances extremely difficult to differentiate the cup seen in atrophy of the optic nerve from that of chronic (simple) glaucoma.

In chronic primary, or *simple, glaucoma*, the onset and progress of the disease are extraordinarily insidious. Simple glaucoma begins gradually and imperceptibly : patients rarely complain of pain, headaches, rainbow lights, etc., and usually only present themselves, in an advanced stage of the disease, for defective vision. On examination the visual acuity will be found reduced ; field contracted, most on nasal side ; tension very slightly plus ; cornea clear ; anterior chamber rather shallow ; pupil a little sluggish ; details of fundus beautifully clear ; disc grey, and cupped. "The lens, on simple inspection, may appear wanting in transparency when it is not really so. The inexperienced observer may well be astonished to find that in a glaucomatous eye he can sometimes see every detail of the fundus with the ophthalmoscope through a crystalline lens, which to the naked eye looks like a cataract almost ready for extraction. Unfortunate mistakes of diagnosis sometimes arise from this cause." (Priestley Smith.) Progress is slow, but practically certain, to blindness.

The three most important and characteristic symptoms of all glaucomas are :—

(*a*) Increased tension.

(*b*) Excavation of disc.

(*c*) Contraction of field, beginning at nasal side.

In tropical and sub-tropical countries glaucoma is seldom seen in its acute form, though sub-acute and chronic cases are common enough.

Operative Treatment.—The treatment of glaucoma by iridectomy, which we owe to the genius of Von Graefe, is one of the greatest triumphs of ophthalmic

surgery. "This was one of the most pregnant discoveries in ophthalmology, and one which will for all time redound to the glory of Von Graefe. We have only to remember that formerly every case of glaucoma invariably led to blindness, and that now, thanks to iridectomy, the majority of glaucoma patients can be cured. How many thousands there are that formerly would have been forced to sink year by year irretrievably into the night of blindness, but who now are saved for vision through Von Graefe's discovery!" (Fuchs.) Von Graefe professed himself unable to explain satisfactorily the reason for the beneficent action of the operation in this disease; and even now it cannot be said to have been demonstrated with absolute certainty, though the investigations of Priestley Smith and Treacher Collins[1] have thrown much light on it.

Iridectomy seems to act in some cases by opening up again a large part of the filtration angle through the removal of the portion of iris blocking it (*vide* Operations: Special glaucoma-iridectomy); in others, especially recent cases, by the general slackening of the eye, due to escape of aqueous, permitting the reopening of the whole filtration angle; in many, including probably all old-standing cases, by its leading to the formation of a permanent corneo-scleral fistula.

De Wecker, quoted by Priestley Smith, relates a case in which a patient on whom an iridectomy had been performed for glaucoma was accustomed to relieve slight recurrences of glaucomatous symptoms by pressing on the eye with his finger—a procedure which caused the visible extrusion of fluid through the corneo-scleral fistula beneath the conjunctiva.

In acute and sub-acute glaucoma the sooner iridectomy is performed the better; a delay of even a few hours may be fatal to vision. Even if only perception of light remain when the patient is first seen, iridectomy may restore vision to a wonderful extent, if the glaucoma attack has not lasted too long.

In simple glaucoma operative interference—sclerotomy

[1] Priestley Smith: Erasmus Wilson Lectures, 1891. Treacher Collins: Hunterian Lectures, 1894.

or iridectomy—is also necessary, though its beneficent effect is not so dramatically marked as in the acute form. We cannot hope to *restore* lost vision in simple glaucoma, for the morbid changes are established when we first see a case, but we can usually arrest the disease and prevent the deterioration of vision going any farther. It must be admitted, however, that in some cases operation appears to have no effect whatever, and in others—fortunately rare—it apparently aggravates the disease. This failure of operation in a certain percentage of simple glaucomas has led some (very few) surgeons to abandon operative measures in this form, and to trust to palliative treatment alone. I believe, however, the vast majority of ophthalmologists are quite in accord in deprecating this attitude. *Sclerotomy* (*vide* Operations) has come into vogue of recent years as a substitute for iridectomy in glaucoma; and from my limited experience of the disease—almost entirely confined to Moorfields—I believe it is more satisfactory than iridectomy in *simple* cases. A very guarded prognosis should always be given as regards result of operation in simple glaucoma.

Priestley Smith advocates "scleral puncture" as an adjunct to iridectomy or sclerotomy (*vide infra*).

A blind painful eye may be relieved by iridectomy, but on the whole it is best removed.

Palliative and Auxiliary Treatment.—Eserine, in cases where it is capable of producing myosis, thins and stretches the iris, obliterates its folds, and, when adhesions are very slight or recent, may thus reopen the filtration angle. It is most useful in cases where immediate operation is refused or is for some reason impracticable, as it keeps the eye quiescent, for a time at all events. In cases of simple glaucoma where the patient declines operation its routine use will undoubtedly postpone the onset of blindness. It is best employed according to the Moorfields formula, combined with cocaine (one grain of the eserine to five of cocaine); of course, cocaine has a mydriatic effect, but in this formula the eserine is in sufficient strength to overcome this. Nitrate of pilocarpine (two grains to the ounce) acts in somewhat similar fashion, besides lowering the general circulation tension.

CATARACT.

"Cataract" is an expression used to denote opacity of the lens, whether the opacity be complete or partial. The blindness or visual defect produced by lens opacities was at one time believed to be due to some effusion *poured down* in front of the lens, and the word cataract came into use as the expression of this belief.

Owing to the youth of the men composing the rank and file of the Army, and the age clause which compels a large majority of officers to retire at or before fifty-five, *senile* cataract—a disease of the decades beyond the fifth—is seldom seen in the Army. Zonular and congenital cataracts are, however, very frequently met with. Our *confrères* in the Indian Medical Service see a very large number of cases of all varieties of cataract amongst the natives, and have unrivalled opportunities for acquiring dexterity in operating.

Partial Cataracts.

Partial cataracts—to which class the congenital and zonular forms alluded to above belong—are those most frequently seen in military practice.

Anterior Polar Cataract.—Anterior polar cataract shows under focal illumination as a small grey spot at or near the anterior pole of the lens. It is most often congenital, and due to some obscure disturbance of nutrition; it is occasionally caused by a perforating central ulcer of the cornea.

Anterior Capsular Cataract.—Here there is a circumscribed opacity at some part of the anterior capsule of the lens, due to adhesion of a persistent pupillary membrane or to a syneclira. Anterior polar cataract is really a variety of this.

Posterior Polar Cataract.—Here there is a small white spot on the posterior pole of the lens; difficult, perhaps, to see by focal illumination, but showing up clearly as a black spot with transmitted light. It is a small capsular cataract, congenital, due to persistence of part of the hyaloid artery.

Anterior and Posterior Cortical Cataract.

—Posterior cortical cataract is essentially a secondary lesion, following advanced fundus disease such as retinitis pigmentosa, etc. It generally assumes a pretty stellate form, the rays of the star curving forward from the posterior pole to the periphery ; or it may look like a delicate grey bowl, into the interior of which the observer looks. Anterior cortical cataract is a similar condition of the anterior lens-cortex ; it is very rare.

Central and Axial Cataracts. — Central cataract appears as a small sharply defined grey spot in the centre of the lens ; it may extend to the anterior and posterior poles, resembling a tiny torpedo, and is then known as "axial" cataract. Both forms are congenital.

Punctate Cataract is another congenital form. Tiny white dots are seen in the lens, the intervening lens substance being clear ; they are often symmetrically arranged, forming pretty figures, or may resemble a shower of snow.

Congenital cataracts are usually bilateral.

Zonular or **Lamellar Cataract** is, I believe, always of congenital origin, though the opacity may be very slight at birth. It differs, however, from other congenital forms in being distinctly, though very slowly,

Fig. 24.—Some Forms of Zonular Cataract. (Liebreich.)

progressive ; it is bilateral, the cataracts seldom differing much in size or density. The special characteristic of zonular cataract is that the *nucleus* is clear, but surrounded by one or more opaque *zones*, which zones have a clear layer of lens outside them. The annexed diagram (from Liebreich) of different forms of zonular

cataract, explains the formation better than words. Zonular cataract is occasionally complicated by axial cataract (*vide* second figure in diagram).

Zonular cataract is best seen with transmitted light in the fully dilated pupil; a dark central opacity, varying in density and diameter, and darker at the periphery than the centre, then shows up clearly against the red background. Little spokes, projecting from the margin of the opacity like the handles of a steering wheel, are believed to be indications that the cataract is actively progressive. Unlike other congenital cataracts, the zonular form is seldom accompanied by amblyopia or other eye defect; so that if the opacity be small normal vision may be obtained through the peripheral clear zone by means of an optical iridectomy, or, with dilated pupil, through a stenopœic slit. In most cases it may for practical purposes be considered stationary, though undoubtedly it slowly increases in density, and the vision at fifty will be found considerably less than it was at ten. Nearly all patients present some evidence of rickets, more especially "rachitic teeth"—characteristic irregularities of the enamel of the central incisors; many give a history of "fits" in infancy. The defect of vision, of course, varies with the density and diameter of the cataract.

The *treatment* of partial cataracts, when treatment is necessary, consists in *discission* (with or without curette evacuation) or iridectomy. The advantage of iridectomy is that the power of accommodating is retained. When a large clear zone surrounds a small central opacity, and the patient is found to see well through dilated pupil and a stenopœic slit, an "optical iridectomy" in each eye gives the best results (*vide* Operations). Discission is unsatisfactory after thirty, owing to the hardness of the lens nucleus and the tendency of the iris to inflame from contact with the lumps of absorbing lens tissue.

Evidences of congenital visual defect, such as nystagmus, as a rule contraindicate operative interference, for the removal of lens or iris will probably have little effect in improving vision.

Complete Cataracts.

Complete congenital cataract is occasionally seen. It is very often accompanied by amblyopia or congenital fundus lesions. Treatment consists in needling.

Senile Cataract.—*Pathogeny.*—As age advances the nucleus of the lens hardens, and the whole lens gradually loses its elasticity, and with it the power of assuming a spherical shape in response to the action of the ciliary muscle: this loss of accommodation becomes so pronounced at forty-five that emmetropes find they have to wear convex glasses for near work at or soon after that age. In certain persons this hardening and general loss of elasticity is accompanied by arrest of growth of the lens fibres, with the result that the fluid in the lens finds its way between the fibres, forming spindle-shaped spaces, while molecular changes take place in the fibres themselves. The progress of these changes produces first opaque striæ, and ultimately total opacity of the lens, or "senile cataract."

Symptoms.—When the whole lens is opaque the grey opacity occupying the pupillary area is easily detected by inspection or focal illumination. In the early stages opacities appear as fine striæ, dots and lines, or faint cloudiness, and are best seen by transmitted light or direct examination with a high *plus* lens. When the opacity has extended throughout the lens the cataract is said to be "mature" or "ripe": there is then no fundus reflex to be seen, and the iris, obliquely illuminated, throws no shadow on the opacity. Within a variable time after maturity the cataract becomes *hypermature*, either shrinking into a flat chalky mass or, more rarely, liquefying in its cortical portion, while the hard brown nucleus sinks to the bottom of the capsule, in which it moves about with the motions of the eye (Morgagnian cataract).

Vision deteriorates *pari passu* with the extension of the opacity. In the very beginning the lens becomes more spherical in shape, so that patients wearing presbyopic glasses find them "too strong" for them, or may even develop myopia (cataractous myopia); polyopia, from unequal refraction, is also often com-

plained of in this stage. As the lens changes progress dots and clouds, moving with the eye movements (thus differing from vitreous opacities), disturb vision; these gradually increase, and, owing to the fact that the opacities causing them generally affect first and most the centre of the lens, patients acquire the habit of seeking dim light or sitting with the light behind them, in order to keep the pupils as much dilated as possible. After a time, only large objects can be seen; and sooner or later vision deteriorates to "hand movement," or even P. L. (perception of light). Even then *projection* —tested by throwing a beam of light on to the eye from various directions and seeing whether the patient can indicate the point from which it comes—should be good unless the case is complicated by fundus lesions. Senile cataract affects both eyes, but seldom begins at the same time in each.

The rate of progression of senile cataract varies greatly, sometimes only a few months, sometimes years, elapsing before maturity is reached. Artificial ripening of cataracts by various means has been tried, but on the whole it is better in cases of necessity to extract an immature cataract, even with the risk of leaving lens substance behind, than to have recourse to any of these measures. As a rule perinuclear cataracts ripen more slowly than peripheral; but no definite answer should ever be given to the question so frequently asked, "How soon will the cataract be ripe?" In cases of slight opacity in one lens, the other being still clear, it is well to postpone informing the patient he has cataract until the advance of the disease renders it necessary, for the very word is apt to excite consternation in the laity; his relatives, however, should be informed, or he may be told he has "striæ in the lens" or "slight lens opacity."

Diabetic Cataract.—Cataract is a frequent complication of diabetes, and is probably mainly due to abstraction of fluid from the lens by the sugar-containing aqueous humour, though the amount of sugar in the latter is extremely small. It is a complete opacity of the lens, commencing in the cortical fibres, and rapidly extending to the whole. Cases of spontaneous absorp-

tion of diabetic cataracts have been reported by Seegen, Koeing, and Tannahill. Extraction in suitable cases is not contra-indicated, though at one time the operation was supposed to be a dangerous one in diabetics, as cases are recorded in which diabetic coma followed.

Treatment.—There is only one treatment for "hard" or senile cataract, viz. extraction of the opaque lens. The old operation of "couching," *i.e.* dislocating the lens into the vitreous by means of a special needle, has long been abandoned.

A caaract should be extracted as soon as it is mature, whether the other lens be affected or not; for, though binocular vision will hardly be practicable owing to the enormous difference between an aphakic and a phakic eye, the eye operated on can be gradually brought into use as the other lens becomes opaque, will be helpful by enlarging the field of vision, and is "a reserve for the future," as Fuchs puts it. Traumatic cataracts and their treatment are discussed in the chapter on Injuries.

After removal of the lens, a powerful convex lens must be worn to take its place in the refraction system of the eye (*vide* Operations).

Secondary Cataracts.—Some varieties of secondary cataracts have been alluded to. Others are produced by deposit of lymph from inflammation of the iris or ciliary body, by detached retina, by glaucoma, etc. The treatment is the treatment of the primary lesion; extraction is seldom of much service, and may be impossible owing to adhesions.

CHAPTER XV.

OPERATIONS.

Ophthalmic operations cannot be taught by book descriptions and rules; the only way in which the necessary ambidexterity and delicate sureness of touch can be attained is by going through a prolonged course of practical instruction with a skilled operator. An excellent means of learning the technique is to practise the various operations on pigs' eyes; the little apparatus for fixing the eyes can be obtained from Messrs. Curry and Paxton, Great Portland Street, for a trifle. Descriptions of operations are mainly useful for purposes of reference when doubt is felt as to the exact sequence of procedure in particular operations, the precise instruments employed, the site of incisions or sutures, etc.

I propose to give in this chapter a very brief description of the chief ophthalmic operations, the bald outlines of which must be filled in by the reader from his own work and experience.

OPERATIONS ON THE PUNCTA AND CANALICULI.

Stenosis of Punctum or Canaliculus.—The punctum is easily seen by everting the lid; the lower is the one usually affected. It will often be sufficient to dilate the punctum and canaliculus with a Lang's or Nettleship's probe; when this proves insufficient they must be slit up by a canaliculus knife, the edge being directed towards the eye.

Stenosis of the Nasal Duct is the chief cause of inflammation or blenorrhœa of the lachrymal sac—a condition in which there is annoying epiphora, with regurgitation from the sac into the eye on pressure, developing in many cases into abscess of the sac. Stenosis may be due to simple catarrhal inflammation of the duct, but is very often the result of syphilitic disease

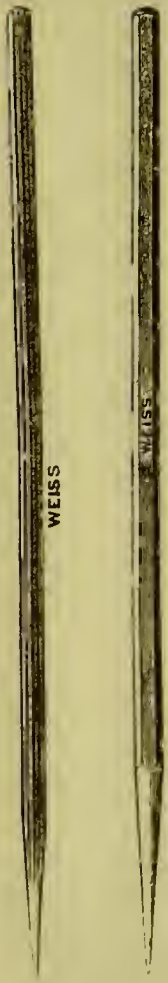


FIG. 25.—PROBES AND CANALICULUS KNIFE.

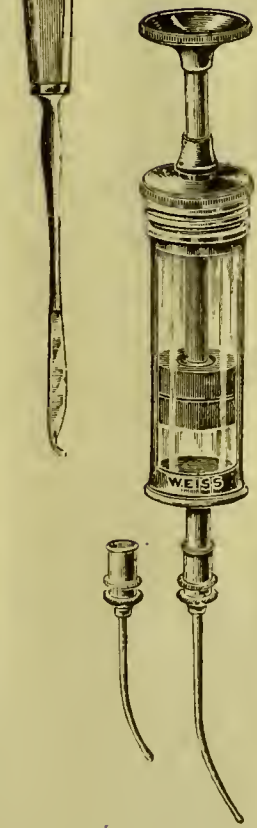


FIG. 26.—LACHRYMAL SYRINGE.

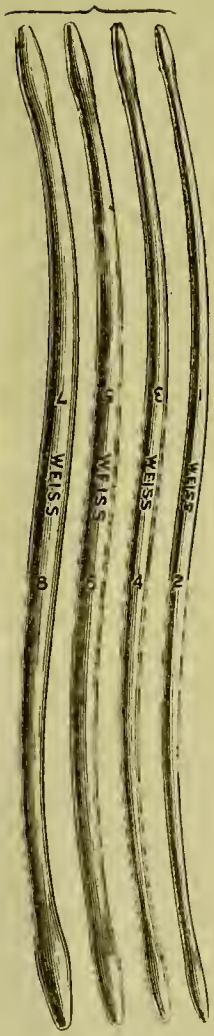


FIG. 27.—COUPER'S PROBES.

—congenital or acquired. The diagnosis of stricture may be rendered certain by syringing through the lower canaliculus; if the fluid regurgitates there is evidently some obstruction to its passage down the nostril.

The treatment is to slit up the lower canaliculus with a canaliculus knife, and then pass a lachrymal probe of the largest possible size down the duct. This is a painful procedure; in children an anæsthetic will be required. The little operation requires practice; it gives immediate relief even in cases in which there is violent dacryocystitis. The probing must be repeated at regular intervals; in obstinate cases it is a very good plan to introduce a "style" of soft metal into the duct, with its upper end bent out over the lower eyelid, and to leave it there for some weeks. Syringing the sac and duct daily with some antiseptic or mildly astringent solution accelerates the cure. When an abscess of the sac is already pointing it must be opened externally, thoroughly washed out and treated with some astringent solution; if at the same time the duct be made thoroughly patent, a fistula rarely forms. When a fistula does remain its edges may be pared and stitched, or touched with a hot wire; in bad chronic cases an attempt may be made to dissect out the sac, but it is a most difficult operation and seldom successful.

OPERATIONS ON THE LIDS.

Ptosis.—Paralytic ptosis may be relieved, apart from the treatment of its cause, by means of a wire support attached to a spectacle frame so arranged as to hold the lid up. Operative treatment is required in congenital ptosis, and in paralytic cases where other treatment has failed.

In mild cases it may suffice to excise a semilunar piece of skin and muscle from the lid, bringing the edges of the wound together with sutures.

The object of *Panas's operation* is to bring the power of the frontalis muscle to bear on the lid.

An incision is made down to the periosteum just below the margin of the orbit, and another, a little longer and through skin only (*a*) just above the eyebrow; the skin and muscle between the two is then undermined. A tongue

of skin and muscle (*s*) is then formed on the lid, pulled up under the bridge, and stitched to the upper lip of the incision (*a*) by the double-needled suture (*b b*). A spatula of horn or tortoiseshell must be passed under the lid in making the lid incisions.

Entropion.—The most annoying symptom produced by entropion is the scratching of the eye by the inturned cilia (distichiasis—trichiasis). Epilation with cilia forceps

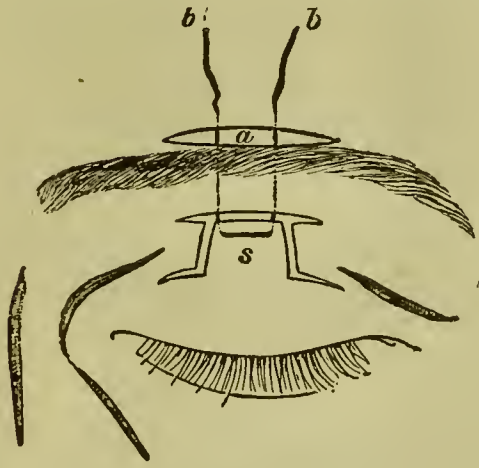


FIG. 28.—PANAS'S OPERATION FOR PTOSIS. (FUCHS.)

gives temporary relief, but, of course, the hairs grow again. Electrolysis may be tried when only a few hairs grow in, but it is a tedious procedure, and by no means certain.

In the *Arlt-Jaesche operation*, the object is to displace the portion of lid from which the hairs grow. The lid is grasped in an entropion forceps, the skin being pressed back so as to show the lid margin. (In the illustration, after Arlt, a horn spatula is used instead of an entropion forceps.) In the lid margin an incision 3 or 4 m.m. deep, just in front of the openings of the Meiboinian glands, splitting the lid along its whole length, is made with a triangular knife or an ordinary cataract knife; the anterior lip of this incision contains the bulbs of the

hairs. A semilunar incision is then made through the skin of the lid throughout its entire length, the lower part parallel to and about 5 m.m. from the margin, and

Fig. 29.—Cilia Forceps.

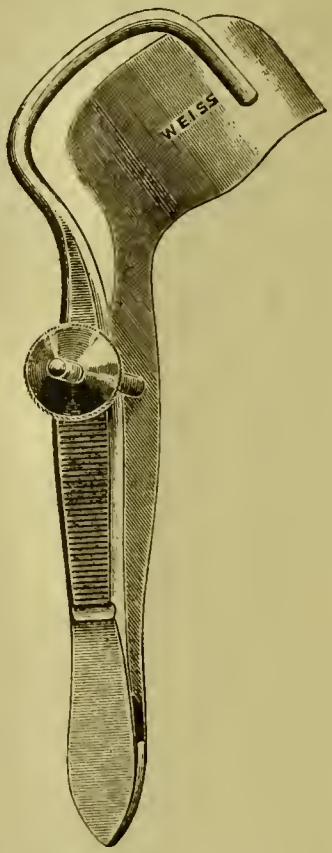


Fig. 29a.—Entropion Forceps.

extending a little beyond the ends of the marginal slit. the upper curved portion joining this at its extremities and curving away about 4 m.m. from it in the centre ; this skin flap is dissected off. The edges of the wound are brought together by sutures which, including only skin

in the upper lip, take in both skin and a little muscle in the lower, so that the lower flap of the wound is drawn up, carrying with it the hairs.

Burow's operation is an excellent one in cases of bad entropion following trachoma, where the tarsus itself is bent inwards. In this operation the lid is everted on a horn spatula, and an incision made through conjunctiva

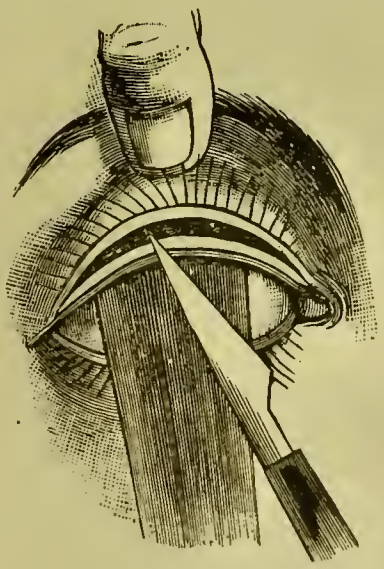


Fig. 30.—After Arlt.

and cartilage, about 3 m.m. from the edge, from end to end of the lid along a whitish line which indicates the point where it is bent. The lid is then turned back and held in an entropion forceps; two sutures are passed under the skin of the upper lid as shown in the diagram, leaving loops in the centre. A small roll of plaster, adhesive side out, is then laid along the lid under the loops, which are tightened over its middle; the free ends of the sutures are knotted over its extremities. In this way the roll of plaster forms a fulcrum by which the divided tarsus is kept everted; after a few days it may be

removed with the sutures. I have seen this operation followed by most excellent results.

In *Snellen's operation* an incision is made through the skin of the lid, 3 m.m. from its border; through this a narrow band of orbicularis is removed, and also a wedge-shaped strip of cartilage. By means of three double-needled sutures the skin containing the cilia is pulled up to the upper edge of the *cartilage* incision, beads being passed down on the threads to prevent their cutting the skin. The diagram, by Wecker, shows the way in which

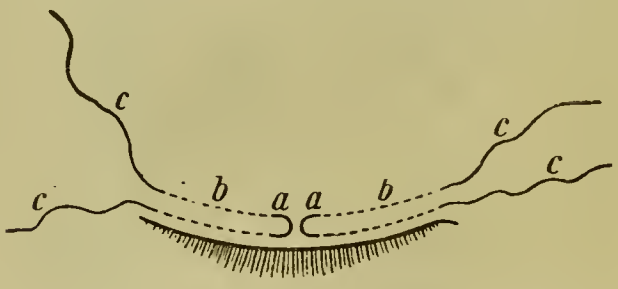


FIG. 31.—BUROW'S OPERATION.

Diagram of upper lid showing direction of sutures: the dotted lines, *b*, *b*, show where they pass under the skin. The roll of plaster is passed through the two central loops, *a*, *a*, and the free ends of the sutures, *c*, *c*, are tied over each extremity.

the sutures act on the grooved cartilage; they may be removed on the fourth day.

Ectropion.—Cases in which the lid is everted through chronic conjunctival swelling are often easily and quickly cured by means of *Snellen's suture*. Strong large curved needles are threaded through each end of a piece of twisted silk; both needles are then entered at the centre, or most bulging part of the everted conjunctiva at a distance of ½ c. m. from each other, passed deeply down, and brought out through the skin 2 c.m. below the lid margin, the points of exit being 1 c.m. from each other. The ends are pulled tightly, with the result that the loop on the lid everts it, and are then tied over a roll of plaster on the cheek. More than one suture may be required. In ectropion due to adherent cicatrices of the skin some modification of the

plastic operation known as the "V.Y." gives the best result. *Arlt's operation* is applicable to ectropion caused by caries of the margin of the orbit. The diagram shows

Fig. 32. (Wecker.)

the incisions. The incisions *a b* and *b c* are through skin and muscle, the whole triangular flap *a b c* being dissected free; the cilia-bearing margin *c d* is excised; *c* is then sutured to *d*, and the space left where the apex of *a b c* was is covered by bringing its edges together as in a V.Y. operation.

Symblepharon.—Cases of adhesion of lid to globe (*vide supra*, Injuries) where the adhesion does not

Fig. 33. (Arlt.)

extend to the bottom of the fornix can be successfully treated by separating the adhering surfaces and keeping them apart until healed. Symblepharon extending right into the fornix is, however, very difficult to cure. An attempt may be made by separating the adhering surfaces, and then covering the bulbar raw surface by stitching the loosened conjunctiva over it. Transplanta-

tion of skin from the upper lid to the inner surface of the lower has also been tried. It must be confessed that operative interference in cases of complete symblepharon is rarely successful.

ENUCLEATION.

A general anæsthetic is required for this and the following operations. In large clinics it is as well to mark the eye it is intended to remove some time prior to the operation, at all events when the eye presents no striking outward change, as cases have actually occurred in which the wrong eye was enucleated.

Having inserted a speculum, the surgeon stands behind the patient and grasps, with forceps held in the left hand, a thick fold of conjunctiva on the right side of and close to the cornea, whether the eye be right or left. A hole is made in this with scissors, which are then made to snip through the conjunctiva completely round the cornea, cutting first the lower, then the upper, semicircle. An assistant then holds up the edge of the conjunctival pocket thus made, while the operator passes a strabismus hook held in the left hand under each muscle, and cuts the muscle across with scissors. When all the muscles are cut, the speculum is pressed gently back and the globe proptoses ; the optic nerve is then easily cut across by scissors passed deeply into the orbit behind the globe ; one or two cuts of the scissors will now free the globe of remaining adhesions. The edges of the conjunctival pocket are then stitched together to form a stump for the artificial eye (*prothesis*), which, however, should not be inserted until some weeks have elapsed.

EVISCERATION (MULES'S OPERATION).

Mules's operation of evisceration is an excellent one in cases where complete removal of the globe is unnecessary ; the sclerotic is left, stitched over a glass ball, thus making a large freely movable stump for the prothesis.

An incision is carefully made in the sclerotic just external to the cornea down to the chorioid ; one end of a slender scissors is introduced, and the sclerotic cut through

slowly and cautiously all round the cornea ; then with a sharp spoon inserted between the chorioid and sclerotic the whole contents of the sclerotic cup are scooped out, anything left behind being scraped away afterwards. The cup left is freely washed out with hot antiseptic solution till bleeding has ceased ; then by means of a special instrument a small glass ball is inserted into it, and the sclerotic very conscientiously stitched over it. The success of the operation depends mainly on two conditions : the glass ball must not be too large, and the sclerotic stitches must be numerous and closely approximated. Failing these precautions the ball is certain to be extruded sooner or later. It is hardly necessary to add that the strictest antisepsis is required.

I have seen Mules's operation repeatedly performed by my old chief, Mr. Treacher Collins, with the best results. The stump is so large and moves so freely, that with a well-fitting glass eye over it the cosmetic effect is marvellous.

Inserting a glass ball into Tenon's capsule, instead of into the sclerotic, has been tried ; but the resulting stump in the few cases I have seen has been too lax, so that it fell on to the lower eyelid.

EXENTERATION.

Exenteration means the removal of the entire contents of the orbit ; it may be necessary in cases of malignant disease, etc. The steps of the operation necessarily vary so much according to the nature of the lesions for which it is performed, that no precise rules can be laid down.

OPERATIONS ON THE CORNEA.

Paracentesis is often an admirable treatment for ulcers threatening to perforate or for "hypopion ulcers" (ulcers accompanied by pus in the anterior chamber) ; in such cases it may be performed with an ordinary needle through the floor of the ulcer. Paracentesis with a keratome is required in "curette evacuation" of cataract, to relieve tension, in cases of ulcer in which the floor is not markedly thinned, etc. The keratome is inserted at right angles to the cornea near the corneo-

scleral margin; as soon as the anterior chamber is entered the handle is depressed until the blade is parallel to the plane of the iris, it is then pushed forward until an incision of the necessary size is made, when it is very slowly withdrawn.

In cases of "Saemisch's ulcer" of the cornea (*ulcus serpens*), a creeping ulcer of most intractable nature, if

FIG. 34.—KERATOME.

atropine, heat, and bandaging fail to cure within three or four days, a special kind of paracentesis should be performed. A narrow Graefe cataract knife should be passed through the anterior chamber behind the ulcer, the entrance and exit being in clear cornea on either side; the blade is then turned forwards and the knife made to cut its way out very slowly through the ulcer. Followed by atropine and a bandage, this little operation often has an almost magical effect in cleaning the ulcer and promoting healing. It is occasionally

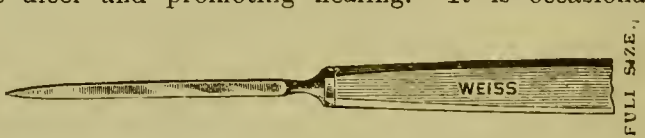


FIG. 35.—NARROW GRAEFE KNIFE.

necessary to keep the wound open for a few days by separating the lips with a probe.

In *keratoconus*, or conical cornea, a condition the cause of which is unknown, the cornea thins and points in the centre in the form of a cone, producing irregular astigmatism and great defect of vision. To procure the flattening of this cone various measures have been tried, chief of which are **removal of a wedge-shaped piece of cornea** and **cauterising the cornea.** In the former operation a wedge-shaped piece of cornea—base on the surface, apex down to or

through Descemet's membrane—is cut out with a very sharp Graefe knife above or below the cone; the healing of the resulting wound will, in successful cases, flatten out the cone. Cauterising the cornea down to, or better still through, Descemet's membrane is also performed for the same condition with fair success; and the same operation is frequently necessary in intractable ulcers. The galvano-cautery is now generally used, the needle being applied cold to the ulcer, heated by making the connection, and then quickly removed. When it is not available a platinum wire heated in a spirit lamp may be employed instead.

OPERATIONS ON THE SCLERA.

Anterior Sclerotomy is nowadays preferred by many surgeons to iridectomy in cases of simple glaucoma (*vide supra*). The operation is not a difficult one, but requires care in its performance to prevent prolapse of the iris. This accident sometimes happens no matter how carefully the sclerotomy is done; when it does it is much better not to attempt to "repose" the prolapsed iris, but simply to draw it out and snip it off, converting the operation into an iridectomy.

The eye being cocainised and a speculum introduced, the surgeon stands behind the patient and fixes the eye by grasping a fold of conjunctiva below the cornea with forceps (held in the right hand for the left eye, and *vice versâ*). A Graefe knife (held in the right hand for the right eye, the left for the left) is then introduced, with the edge towards the operator, into the anterior chamber through a point in the sclera 1 m.m. behind the corneal margin and just above the horizontal meridian, and brought out at the same point on the opposite side. (Care must be taken that the edge is directed *towards* the surgeon; in Weiss's knives a small dot on the back of the handle indicates the side corresponding to the back of the knife.) With a sawing motion the sclera is now cut upwards till a bridge 3 m.m. broad is left, when the knife is slowly withdrawn. Should

FIG. 36.—DIAGRAM SHOWING INCISIONS IN SCLEROTOMY.

the iris become entangled in the knife in the course of the operation—an accident made evident by the pupil assuming an oval contour—the best thing to do is to withdraw the knife at once and do an iridectomy. On completing the operation eserine is instilled, and the eye bandaged.

Posterior Sclerotomy.—Mr. Priestley Smith considers posterior sclerotomy ("scleral puncture") a useful adjunct to a glaucoma iridectomy, performed immediately before the latter at the same sitting. Other distinguished operators bear testimony to the efficacy of this combined operation, but it can hardly be said to have come as yet into general use.

"The patient turns the eye inward, so as to expose the outer part of the sclera, and looks steadily at a lighted candle placed in the requisite position. The surgeon, taking a Graefe knife in one hand and a forceps in the other, seizes the conjunctiva near the horizontal meridian and slides it downwards a little over the sclera. He then punctures the sclera on the horizontal meridian at a point at least 5 m.m. from the margin of the cornea, keeping the back of the knife towards the cornea and the point directed towards the centre of the globe so as to avoid touching the posterior pole of the lens. After entering about 10 m.m. the knife is slowly withdrawn, and at the same time slightly rotated on its axis, so as to give a gaping wound through which fluid or consistent vitreous escapes. The conjunctiva is then allowed to slide back into its place." (Priestley Smith.)

OPERATIONS ON THE IRIS.

Optical Iridectomy.—The object of an optical iridectomy is to enable a patient with some central opacity—such as corneal cicatrix or partial cataract—to see through an artificial coloboma. The coloboma must be narrow, and, if possible, down and in. After cocainisation, introduction of a speculum, and fixation, a keratome is introduced into the anterior chamber through the margin of the cornea at the desired spot, and then slowly withdrawn. An iris forceps is now passed in, blades closed, right to the edge of the pupil, then opened a very little, and closed again on a fold of iris, which is drawn

out and snipped off close to the wound by iris scissors (I prefer a small plain pair of scissors curved on the flat) pressed close to the globe before cutting. Any portion of iris prolapsed into the wound must be gently reposed. The greatest care must be taken to avoid injuring the lens.

Glaucoma Iridectomy. — The iridectomy is usually done upwards. The keratome may be employed for opening the anterior chamber, but the latter is so shallow in glaucoma cases that it is better to make a small corneal flap with a narrow Graefe knife entered at the limbus, brought out at a similar point on the opposite side, and made to cut its way out upwards through the limbus. Even when the incision is made in this way, it may be very difficult in cases with very shallow anterior chambers to get into the chamber and at the same time avoid the iris. Often the operator fancies his knife is in the chamber when it is really in the substance of the cornea —an accident indicated by wrinkling of the cornea and absence of aqueous outflow. Having opened the anterior chamber, the iris forceps is introduced closed, opened, and closed again on a fold of iris, which is then drawn out to its fullest extent. The fold so drawn out is now *nicked* only by the scissors close down at one corner of the wound, then *pulled* over to the other corner so as to *tear* it out and so remove as much of the angle-blocking part as possible; finally the remaining tag is snipped across. "Reposing," and "toilette" of wound, as usual.

FIG. 37. — IRIS FORCEPS.

Iridotomy.—This simply means making a hole in a closed iris so as to restore communication between the chambers. It is sometimes necessary in cases where iritis and occlusion of the pupil have followed cataract extraction ; obviously the operation would be inadmis-

sible in a phakic eye. A narrow Graefe knife may be passed right through the anterior chamber into the centre of the iris or pupillary membrane ; or a cross-shaped hole in the iris may be snipped by a De Wecker's scissors passed in through a keratome opening. The occasion for iridotomy seldom arises.

Iridodialysis.—De Wecker performs an operation for glaucoma—"combined sclerotomy"—which consists in introducing forceps through a scleral incision, seizing the iris near its *periphery*, and pulling it towards the pupil till its base is torn away, thus producing iridodialysis. I have never seen this operation.

OPERATIONS FOR CATARACT.

Discission or Needling is employed in "soft" cataracts, *i.e.* in the young ; to procure the absorption of non-cataractous lenses in myopia ; and to tear holes for

Fig. 38.

optical purposes in remains of capsule left after cataract extraction. The operation is very simple. The pupil having been dilated by atropine, the eye is cocainised and fixed ; a discission needle is then passed through the margin of the cornea into the lens in the pupillary area, where with a movement or two it is made to tear the lens capsule. (Most needles have a "stop" to prevent their being passed too far in.) The needle is then withdrawn quickly. Sometimes two needles are passed in, one from either side of the cornea to the same point in the capsule ; by manœuvring the handles large tears can thus be made. This double needling is most suitable in cases where tough capsule has been left after cataract extraction.

After needling, the lens swells up and becomes opaque, to be subsequently gradually absorbed. This process of absorption takes weeks or months, and needling has often to be repeated. In cases of bilateral cataract or in myopia it is usual to needle one lens, and wait for its absorption or evacuation before doing the other.

Curette Evacuation soon after needling is now pretty generally practised, as the process of absorption is so slow. In some needling cases the suddenly swelling lens produces *plus* tension or iritis, and evacuation is obligatory. A corneal flap is made with a narrow Graefe knife, as in iridectomy; then by depressing the upper lip of the incision with a curette, and at the same time gently stroking the cornea upwards, the opaque lens masses are evacuated. In the old *suction* operation the nozzle of an aspirator was introduced through the corneal incision, and the lens sucked out; but as the soft lens matter flows away quite easily it is difficult to see where the utility of suction lay.

Extraction.—Before operating, the conjunctiva and lachrymal apparatus are carefully examined to see that they are free from any discharge likely to cause sepsis. The cataract itself should be mature or "ripe," unless for some special reason it is considered necessary to extract one not quite mature; the patient's "projection" is also tested (*vide supra*). Should he suffer from severe cough, it is as well to postpone operating for a little time.

The eye is cocainised and irrigated thoroughly with warm antiseptic solution. The operator stands behind the patient, and, having inserted a speculum, fixes the eye by grasping a fold of conjunctiva below the cornea with forceps held in the right hand for the left eye, and *vice versâ*.

First Stage.—Opening the Anterior Chamber.—A Graefe knife, held in the right hand for the right eye, the left for the left, is entered at a point very slightly external to the corneal limbus and just above the horizontal meridian, passed across the anterior chamber, and brought out at the same point on the opposite side; care must be taken that the edge of the knife is directed towards the operator before it is inserted. With two or three long sawing strokes of the knife the limbus is cut upwards until the top of the cornea is reached; then the last little bridge is cautiously cut across, and the tag of conjunctiva left turned down on the cornea. The knife and fixation forceps are now laid aside, and *the speculum removed*—great care being taken to avoid pressing on the globe in doing so.

Second Stage.—Iridectomy.—The patient is told to look down. The operator, holding up the lid with the left little finger, passes an iris forceps held in the same hand into the anterior chamber, draws out the iris, and snips it off close up to the incision with scissors. Iris forceps and scissors are then laid aside.

FIG. 39.—DIAGRAM OF CORNEA, SHOWING ENTRANCE AND EXIT OF KNIFE, AND LINE OF INCISION.

Third Stage. — Capsulotomy. — The patient still looks down ; the lid is held up by the operator's left forefinger. A combined cystotome and curette is taken in the right hand ; the cystotome is passed into the anterior chamber with its point sideways until it reaches the lens, when it is turned down and made to score three rents arranged triangle fashion in the capsule. It is then withdrawn, and the instrument turned round in the hand so as to have the curette ready for the next stage.

Fourth Stage.—Delivery of Lens.—The lower part of the cornea is gently pressed on by the convex side of a cataract spoon till the lens tilts forward and up to the

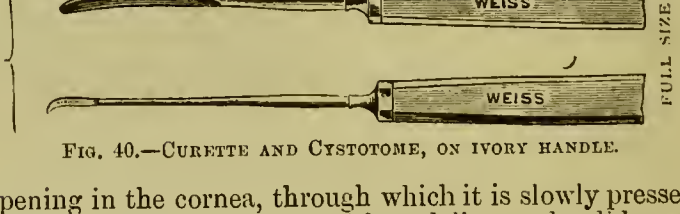


FIG. 40.—CURETTE AND CYSTOTOME, ON IVORY HANDLE.

opening in the cornea, through which it is slowly pressed by the following spoon. After delivery the lids are closed gently for a few seconds, then opened.

Fifth Stage.—"Toilette."—The cornea is gently stroked with the spoon from below up, to clear out any remaining lens substance. The wound is washed with antiseptic solution trickled over it, the iris "reposed," and the tiny conjunctival flap turned over.

Finally the lids are closed, a strip of gauze soaked in antiseptic solution laid over them, and over this a soft pad of antiseptic wool filling the hollow of the orbit ; a similar pad is placed over the other eye, and both retained by a bandage. Patients must be strictly

warned to avoid touching the dressings, and it is advisable to tie the hands at night.

Many distinguished operators leave out the second stage, *i.e.* extract without iridectomy. The cosmetic result in successful cases is excellent, but there is undoubtedly great danger of prolapse of the iris.

Accidents During the Operation.—There may be *difficulty in delivering the lens.* This is sometimes caused by the capsule of the lens not having been sufficiently incised—in which case the cystotome is made to score the edge of the lens—but is more frequently due to insufficient size of the opening into the anterior chamber. Trying to force a lens through an incision which is too small is a dangerous practice ; it is better to enlarge the incision on each side with small curved scissors at once, and then

FIG. 41.—CATARACT SPOON.

deliver. *Prolapse of vitreous* before the delivery of the lens is an unpleasant and far from uncommon accident ; the object of removing the speculum so early in the operation is to prevent this. It is generally due to the operator pressing too hard in attempting delivery of the lens ; sometimes, however, a weak zonula renders prolapse unavoidable. When the ominous viscid bead is seen, pressure on the cornea must be at once stopped, and the lens removed by means of a spoon passed into the anterior chamber under it. As a rule no difficulty is experienced in taking it out in this way, but unfortunate cases occur every now and then in which it sinks deeply into the vitreous and cannot be got out. *Version of the lens* during the fourth stage is rare. I have only seen this accident once ; in that case the lens turned completely over three times before its delivery was finally effected. A bad accident, fatal to sight, is *chorioidal hæmorrhage* after delivery ; a pressure bandage is the only treatment. I have never seen a case.

After Treatment.—The patient, retaining the horizontal position, is carried or wheeled off to a darkened bedroom as soon as the eyes have been bandaged ; it is as well to give a sedative draught immediately after the

operation, as considerable pain is often felt in the eye for some hours.

Unless great pain be complained of, it is better to avoid meddling with the eye for thirty-six, or even forty-eight, hours. The first inspection should be simply a "peep" at the eye, the light being held well to the side; if there be no prolapse of iris and if the eye be otherwise quiet, some atropine may be then instilled, and the eye bandaged up again. If there be a prolapse —a very common accident in extraction without iridectomy—it is best to abscise it at once. For the next three days the eye should be inspected and dressed daily. On the sixth day a simple dry dressing is applied, and left untouched for three days, when it is changed; on the ninth day the patient may be allowed up in a dimly lighted room with the sound eye uncovered. About a fortnight after the operation all dressings can be removed and dark goggles substituted; a day or two later the patient may be allowed out of doors, and he may now be tested for glasses. About + 10 or + 11 D will be needed for distance, + 15 or + 16 D for near work; there is always some astigmatism, so that cylinders may have to be combined with the spheres. The patient must still be seen regularly for a few days longer, and must live very quietly for weeks.

Grey lines on the cornea ("striped keratitis"), due to wrinkling of its deeper layers, are sometimes seen shortly after operation in cases in which the corneal flap has been pulled on a good deal. It almost invariably disappears within a few days. *Iritis* is generally due to some lens tissue left behind; it must be treated on general principles. It occasionally occludes the pupil (*vide supra*, Iridotomy). *Wound infection* occurs every now and then in this operation, even with the most careful operators. Cauterisation may be of service but prognosis is very bad. Iodoform introduced into the anterior chamber is advocated by Römer. *Cystoid cicatrix*, due to incarceration of iris or capsule in the wound, is generally due to careless management of the "toilette" stage; cauterisation is probably the best treatment. *Sympathetic ophthalmia* is a most unfortunate, but luckily very rare, result. *Transient erythropsia* (red vision) and *secondary glaucoma* are other rare sequelæ.

APPENDIX.

VISION REGULATIONS OF THE ENGLISH ARMY COMPARED WITH THOSE OF OTHER ARMIES.

ENGLAND.

(*b*) Candidates for Commissions.

The regulations regarding the examination of eyesight are as follows (Army Orders, July 1st, 1901) :—

"1. If a candidate can read D = 6 at 6 metres (20 English feet) and D = 0·6 at any distance selected by himself, with each eye without glasses, he will be considered *fit*.

"2. If a candidate can only read D = 24 at 6 metres with each eye without glasses, his visual deficiency being due to faulty refraction which can be corrected by glasses which enable him to read D = 6 at 6 metres with one eye, and D = 12 at the same distance with the other eye, and can also read D 0·8 with each eye without glasses at any distance selected by himself, he will be considered *fit*.

"3. If a candidate cannot read D = 24 at 6 metres with each eye without glasses, notwithstanding he can read D = 0·6, he will be considered unfit.

"Normal vision of one eye may be sufficient to allow a higher defect in the other, to the extent of one-sixth, if the defect is a simple error in refraction remedied by glasses.

"The candidate must be able to read the tests without hesitation in ordinary daylight.

"(N.B.—Snellen's test types will be used for determining the acuteness of vision.)

"Squint, inability to distinguish the principal colours, or any morbid conditions subject to the risk of aggravation or recurrence in either eye will cause the rejection of a candidate."

The examination is conducted by a Board of Medical Officers, one of whom possesses special qualifications in vision-testing.

(*b*) **Recruits.**

"In examining a recruit's vision he will be placed with his back to the light, and made to count the dots and describe their position at the distances specified on the test dot card, first with both eyes, and then with each separately. A recruit whose vision has been tested and pronounced good on a primary examination will not, through his own declared inability to see the test dots on secondary examination, be rejected, unless the approving medical officer is satisfied that the man's vision is really defective, and no deception is being practised on him."

The examination is conducted by one medical officer.

FRANCE.

Visual Acuity.

1. For the regular army a *binocular* visual acuity of at least half the normal, $\frac{6}{12}$, without glasses, except in cases of myopia, is required. Monocular v.a. must not, in either eye, be below one-tenth the normal $\left(\frac{6}{60}\right)$.

2. For the auxiliary service men with v.a. in one eye between a half and a fourth the normal, $\frac{6}{12}$ to $\frac{6}{24}$, provided the v.a. in the other be not less than a tenth, $\frac{6}{60}$, will be accepted. Here also glasses are only permitted to myopes.

Visual acuity below these limits causes exemption from military service.

Myopia.

Myopia involves exemption :—

1. When the amount exceeds 6 D.

2. When, the amount being 6 D or less, correcting lenses do not bring the v.a. within the limits laid down above.

3. But myopes above 6 D may be accepted for the auxiliary service if vision can be brought by correcting lenses to the standard indicated in paragraph 2 of the v.a. regulations, provided there be no extensive chorioidal lesions.

Hypermetropia and Astigmatism

Involve exemption from the regular and auxiliary services when they produce defective vision below the limits fixed in paragraphs 1 and 2 of the v.a. regulations.

The regulations also include descriptions of several tests for malingering, and a long list of disqualifying eye diseases. I prefer our own system of leaving the question of disqualifying disease to the discretion of medical officers; otherwise the French rules appear to me excellent in every way, and the standard of vision is a very common-sense one.

AUSTRIA.

The army vision regulations are very lengthy and diffuse; only a very brief summary can be given here. I omit the references to disqualifying *disease*, apart from refraction errors.

By means of the ordinary vision tests, supplemented by ophthalmoscopic examination if necessary, recruits with defective vision are divided into groups to be accepted or rejected in accordance with the following classification :—

A. **Defects which do not relieve from full liability to military service.**

1. Imperfect sight, provided that sight is not less than $\frac{6}{12}$ in either eye, after correction *for short sight*.

2. Squinting, when the sight of the squinting eye is not less than $\frac{6}{12}$, and that of the other not less than $\frac{6}{8}$.

3. Short sight, with focal distance of not less than 10 inches ($= 4$ D). In the case of one-year volunteers the minimum is 8 inches ($= 5$ D); assistant surgeons, apothecaries, and farriers may be accepted with minimum of 6 inches ($= 6{\cdot}75$ D nearly).

4. Long sight (hypermetropia) so long as the strength of the (unaided) sight is not below the minimum fixed in A 1.

B. **Defects which do not relieve from service in the Reserve ("Ersatz-reserve") as "imperfectly fit."**

1. Imperfect sight, not less than $\frac{6}{12}$ with one and $\frac{6}{24}$ with the other (corrected in case of short sight).

2. Squinting, when the sight of the squinting eye is not less than $\frac{6}{24}$, and that of the other not less than $\frac{6}{12}$.

3. Short sight, down to a focal distance of 8 inches ($= 5$ D).

4. Long sight, so long as the v.a. is not below B 1.

C. **Defects relieving from liability to military service except under exceptional circumstances.**

1. Imperfect sight when the v.a. is only $\frac{6}{36}$ with the better eye.

2. Blindness or loss of one eye, even when the other is sound.

3. Short sight, with a focal distance of less than 8 inches (5 D) except in the case of special exemptions mentioned in A.

4. Long sight when the v.a. is only $\frac{6}{36}$ with the better eye.

Imperfect sight when v.a. is less than $\frac{6}{36}$ with the better eye absolutely exempts from service.

GERMANY.

The army vision regulations resemble those of Austria in dividing men of defective vision into groups, but differ in details.

A. **Defects which do not relieve from full liability to military service.**

1. Imperfect sight, so long as the v.a. is above half normal, $\frac{6}{12}$, after correcting any faults of refraction.

2. Short sight, with a focal distance greater than 6 inches (6·75 D nearly), provided the corrected sight be above half normal, $\frac{6}{12}$.

B. **Defect which does not relieve from service in the Reserve ("Ersatz-reserve").**

Squinting, when the margin of the cornea of one eye touches the inner or outer angle of the eyelids, while the other eye is directed straight to the front.

C. **Defects rendering unfit for Standing Army or Reserve, but not necessarily unfit for the Landsturm.**

1. Imperfect sight of both eyes, when the sight is only half normal, $\frac{6}{12}$, or less than half but better than a quarter, $\frac{6}{24}$; to be estimated after correcting any faults of refraction.

2. Short sight, when the focal distance of the better eye is 6 inches or less, but corrected sight above one quarter normal, $\frac{6}{24}$.

3. Blindness of one eye.

D. **Defects rendering permanently unfit for Standing Army, Reserve, and also in general for Landsturm.**

1. Imperfect sight, where sight of the better eye is only one-quarter normal $\frac{6}{24}$, or less.

2. Blindness of both eyes, or blindness of one and imperfect or limited vision with the other.

Colour Vision.

"Men destined for railway troops, balloon sections, and transport will be tested for ability to distinguish colours by the use of woollen test samples or coloured slips. Railway troops must be able to distinguish the colours red, green, and white."

ITALY.

Defective sight is a cause for rejection when (with glasses) the power of either eye is less than one-twelfth normal, $\frac{5}{60}$, or if the power of vision of both eyes is less than one-third of the normal. M. of 6 D or over, or when, with suitable correcting glasses, vision is below the minimum, causes rejection.

UNITED STATES.

The only vision regulations I have been able to find in the "Manual for the Medical Department of the United States Army, 1899," are the following. It will be observed that the old types, in which distance was reckoned in *feet*, are employed.

"*Examination of Cadets and Cadet Candidates.*

"(*b*) Vision, as determined by the official test types, must not fall below $\frac{15}{20}$ in either eye, and not below $\frac{20}{20}$ with both, unless it can be made normal by proper glasses.

"(*c*) Colour blindness is not a cause for rejection, but must be noted upon the form for physical examination, and the applicant so informed."

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